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THE EDUCATION AND FUNCTION OF THE INDUSTRIAL HEALTH TEAM*

BY

RONALD E. LANE

The last 100 years have been remarkable for the technological advances which have changed industrial and domestic life. While these changes have raised standards of living, they have also thrown up new medical and social problems which confront us both as citizens and as doctors. Some of these important medical and social problems are the direct result of the circumstances of occupation, and that branch of medicine which deals with them is called "occupational" or "industrial" medicine. Health cannot be narrowly confined. The doctor must be concerned with the whole life. The field, however, is so vast that it can only be studied in depth if it is suitably broken down. Occupational medicine is concerned not only with the prevention and treatment of industrial accidents and diseases, it includes an appraisal of the many physiological and psychological factors of work. Many complex and difficult problems are involved and their solution calls for the close cooperation of the educationist, the social scientist, and the economist, as well as those whose basic training has been in the medical sciences.

Occupational medicine differs from some other branches of medicine in that its successful practice depends on the sanctions of others. It can achieve little without the cooperation of employer and employed. Both management and workers must understand the purpose of an industrial health service, and the respective roles they play in promoting health in an industrial community. The education of the two main parties in industry must therefore be of considerable importance.

Management

Good management, by preventing unnecessary stress, is in itself an important factor in the health of those comprising an occupational group, and the institution of training for management is a progressive step, and one likely to lead to better health in industry. In bringing order to this training the British Institute of Management is doing a great service. The Urwick Report in 1947 showed that a common basic course was desirable for the various sections of management. It is to be hoped that in any such basic course general occupational health problems, and management's part in their solution, will be emphasized.

The attitude of good management to industrial health should be critical but receptive, free from sentimentality but conscious of responsibility. Such an attitude is only possible where management is informed.

The addition of fundamental knowledge in the occupational health field is the primary concern of research departments. Its application is the responsibility of industry. This knowledge, however, can be applied only by those who are conversant with the practical working of industry. If, for instance, it is established in the laboratory that certain machine designs are less dangerous and less fatiguing than others, it becomes the responsibility of industry to apply that knowledge, not only for its greater efficiency, but also for the greater comfort and safety of the workers involved. Top management should realize that it has such responsibilities, and delegate them to those with the ability to carry them out.

One of the most important changes in Great Britain in recent years has been the greater value placed on the individual as a human being. In industry this has meant a keener appreciation of the importance of human relationships in the working group. This change followed naturally upon the recognition of the need to safeguard the physical side of the working environment. Important as the subject of human relations is, it is one that does not lend itself easily to research, and new techniques are badly needed. These are being slowly evolved. In the small units personal contacts between master and man are easy and relationships, whether good or bad, are at any rate understandable to the workman. In much modern industry the units are too large for this close contact, and the impersonal atmosphere that results is apt to bewilder him. In such units the need for a

* The Mackenzie Industrial Health Lecture (June, 1950).

specialist to deal with personnel problems was soon appreciated. Much good work, based largely on empiricism, has been done for industry by the personnel manager. His work is difficult and depends in no small measure on personal qualities, but there is no doubt that a broad education and wide sympathies are an aid to success. Training has for some time been given for this work, and it is important that it should be broad and practical. In the somewhat nebulous field of "human relations" it is important that the personnel manager should be able to distinguish authoritative new work from the loose pseudo-scientific writing that appears in such profusion. For this a sound background is very valuable, for in this field the value of scholarship and wisdom far outweigh that of technical knowledge.

The Foreman

The personnel manager must not be interposed between the workman and his foreman, for this relationship is of vital importance to the morale of a working group. The foreman holds a difficult post and often needs help with the problems involved in handling groups of people. This has been widely recognized and many courses for foremen pay particular attention to this aspect. This is an important development and one which should be widely extended.

The Workman

Josiah Stamp said that education had three main aims. It should enable those who receive it to get a living, live a life, and mould a world. The education of the workman may be briefly reviewed under these heads.

Industry is keenly alive to the need for training young people, and there is wide provision for promising youngsters on the technical side. It may be that the vocational side is over-catered for, because the tendency to break down complex skilled jobs into routine repetitive operations means that there is not enough skilled work for all those capable of taking the training. Our modern industrial methods tend to eliminate work satisfaction for certain people, and it must be the responsibility of educators to provide for these a substitute. This may be found in the work, or elsewhere.

Certain pioneer firms have led the way in providing continued part time general education up to the age of 18. Often it has proved difficult to avoid making this continued education too technical, since the youngsters themselves are apt to resent training which they regard as having no immediate practical application. This, however, is a matter for skilful teaching, which must satisfy the adolescent need for understanding his place in the adult world, and his emotional relations and for developing his independence of parents. When the county college scheme becomes operative these facilities will be available for all young persons, as well as for the favoured few who happen to work for progressive firms.

The Education Act of 1944 not only envisages the part time education of young people up to 18, but also continued education facilities throughout adult life. These are little more than dreams at present, though the blueprint is there. Such a scheme might provide an outlet and a satisfaction for those who find the unrelieved drabness of monotonous work insufferable. Education could lead to a better use of the leisure which is being made so much more abundant by our technical advances, and by so improving the quality of life it can raise the standard of health. We cannot escape the fact, however, that the absence of work satisfaction in much of present day industry is one of our major industrial health problems.

The increasing responsibilities of the workman in the conduct of industry can only be successfully undertaken if he has a fuller education, a particular requirement of the trade union official and the shop steward. Such education should lay particular emphasis upon his own responsibility for his health. Without his cooperation no safety measures will be truly effective, and the more willing and informed this cooperation in any health programme, the more effective will it be.

Medical Personnel

The training of all doctors, and those who are to work in the industrial health services must be considered.

General Medical Training.—It has been quite unrealistic to divorce the teaching of medicine from a consideration of the environment of the patient, but such has too often been the case in the past. Following the recommendation of the General Medical Council (1936), however, attempts to show the medical student something of the part played by social and occupational factors have been made in many of our medical schools over the last 14 years. These have met with varying degrees of success.

The Goodenough Report (Report of the Interdepartmental Committee on Medical Schools) published in 1944, regarded "the promotion of health as the primary duty of the doctor". It urged that the foundation of training in this subject should be laid in pre-clinical days and that during the clinical

period the student's training should include personal investigation of social and industrial conditions.

The Royal College of Physicians Committee on Social and Preventive Medicine (1943) recommended that all medical schools should recognize the importance of problems associated with industrial medicine, but wisely went on to emphasize that " the medical student is not expected to become an expert in occupational diseases. It is much more important that he should acquire knowledge and understanding of the social problems relating to industry and be able to recognize the mental and physical response of his patient to fatigue, overwork, monotony, and unsuitable hours or type of employment. For this purpose the practical course in preventive medicine should bring him into personal contact with industrial conditions ".

It will be seen, therefore, that during the second world war the bodies influencing the teaching in our medical schools recognized the importance of paying due attention to occupation as a factor in general, preventive, and social medicine, and in 1947 the General Medical Council endorsed these opinions.

A later expression of opinion in Great Britain comes from the Medical Curriculum Committee of the British Medical Association (1948), which again emphasizes the importance of regarding the patient and his environment as a whole. It too points out that instruction in the preventive aspect of disease should be given emphasis by all teachers throughout the clinical period. It opposes the teaching of industrial medicine to the undergraduate as an independent subject, but recommends its inclusion as a special aspect of social medicine. It recommends that examiners in general medicine should expect the student throughout his answers to have regard to social, occupational, and economic factors in illness.

In those medical schools where a department of occupational health exists, however, there are special opportunities, and these should be used for experiment in teaching method. The objects of this teaching must be kept clearly in mind. It should be directed to help the future doctor to understand the society in which he lives. He must learn to recognize the mental and physical responses of the individual to occupational factors. He should have a proper appreciation, not only of the harm associated with certain occupations, but also with the therapeutic value of work and the important part it plays in the treatment of the patient. All this is difficult. It must be remembered that the medical student is not very interested in health, his interest is rightly in diseases. He is learning to be a doctor, and this entails a knowledge of recognized departures from health. It may be held that this is impossible without first having a very clear conception of health. This is attempted during the pre-clinical period, but although anatomy and physiology are taught in detail, health remains ill-defined. Disease, on the other hand, is tangible and more readily capable of demonstration and it naturally remains the first concern of our medical schools. The doctor's basic training must make him familiar with a general picture of disease processes. Already the demands made on the medical student are very heavy and no further overloading of the curriculum is to be countenanced. All that is possible, until some pruning has taken place, is that the effect of home and occupation, and of leisure, on the patient shall be remembered by teachers throughout the undergraduate training, and shall be demonstrated regularly as occasion permits.

The department of occupational health will be most successful if it can influence teachers in charge of some of the older departments, and arouse the enthusiasm of young demonstrators and clinical teachers. It is through these teachers who are in daily contact with the student that the most useful and effective training in this field can be achieved.

Although most of the student's instruction will be given while he sees patients in the wards and out-patient departments, he must see for himself something of industrial conditions. For this the collaboration of industry is essential. I have been deeply impressed by the very ready help I have continued to receive on all sides in this respect, and I am convinced that industry will gain handsome dividends from such a policy. How often in recent years have we heard an outcry from industry about doctors' certificates? The industrialist has tried to enlighten the doctor by inviting him, busy and overworked as he is, to visit the factory and see for himself conditions of work, and then has been deeply disappointed at the meagre response to his invitation. Seeds must be planted in the undergraduate days and carefully nurtured during the whole clinical training.

Special Training for Medical Personnel Entering Industry

Doctors.—The advance of the subject of occupational health ultimately depends on the quality of those who are attracted to it, on their imaginative approach and on their sound basic training. Occupational health is based on clinical medicine, on the social sciences and on what Ryle called social pathology. A knowledge of the social sciences will make possible a better understanding of the structure of society and of the dynamics of our way of life, and social pathology makes possible

scientific study of the quantity and cause of disease and methods of its prevention. The young doctor who is going to work whole time in industry should receive some special training. This was the object of instituting the Diploma in Industrial Health, which was so strongly advocated by the Factory Department of the Ministry of Labour during the last war, and also by the Association of Industrial Medical Officers (1945). The Diploma in Industrial Health has been in existence now for four years and it is perhaps time to reconsider its value. A diploma is granted by two bodies in England and two in Scotland. For three of these diplomas an academic year's training is required. For one, only practical experience in the field is insisted upon. So far 145 diplomas in all have been granted. What has been achieved? A body of people have received training, and it is to be hoped that their contribution to industrial medicine has been better in consequence and that they have been able more quickly to apply sound principles in the industrial communities in which they have found themselves than their predecessors, who, speaking from experience, had to flounder and learn for themselves and often waste several years before achieving a level of efficiency of real value to their industrial community.

Appropriate training of this nature before undertaking responsible work in industry would be regarded by most people as desirable, yet it is surprising how many large firms do not worry about this and are prepared to accept any doctor as being of equal value to them. Until industry demands well trained men and women it will continue to build up its health services on uncertain foundations. A number of men will, in time and after trial and error, overcome the handicap of inexperience; others will find it impossible. But industry is usually wide awake to its interests, and we must ask ourselves why this state of affairs exists. In part it is because industry is unaware that special training is available, or indeed necessary. As teachers we must ask ourselves if the training programmes offered are good enough to attract the better graduates. We must look critically at both our teaching and the syllabuses demanded by the examining bodies. We must see that those who hold diplomas in industrial health are fully capable of meeting the medical demands of industry. Industry, for the most part, is neither familiar with the full scope of industrial health services nor with their contribution to its competitive efficiency. It is for this reason that the trained man is so essential.

The D.I.H. should be regarded as ensuring a *minimum* standard of knowledge. Its mere possession should not entitle the holder to specialist or consultant status. Such a status will depend on additional training and experience, usually in the practical field of industry. The standard of the four examinations for the D.I.H. has been maintained at a uniform level throughout the country, and there is advantage in this in our present state of development. Such a policy is not without danger, however; it would be disastrous if it became rigid and unimaginative, and the time has now come for bolder experiment.

It is essential that training in occupational health should be neither stereotyped nor too elementary; what is needed is an imaginative treatment of general principles of preventive medicine, plentifully illustrated with examples from the best current industrial practice. Here lies the great responsibility of those entrusted with the teaching of the subject in this formative stage. The teachers must keep before them a clear idea of the function of the doctor in industry; he is not there to deal with the end results of faulty environmental conditions, but to anticipate trouble and to prevent it. He is to practise medicine among a "normal" group; he will have the opportunity of detecting the earliest departures from health and of observing the effects upon people of their occupational environment. He will be concerned with questions of morale, and with the medical manifestations of low morale.

There is much room for experiment in all medical teaching, and this applies particularly to post-graduate education. It must not consist of handing out to the student secondhand information, but rather of teaching him to tackle problems for himself. Ample opportunity for practical work must be given and full use must be made of the historical approach. It is of the greatest importance that others besides medical people be brought in to the discussions: social psychologists, engineers, and industrialists, for it is only by widening our horizons that doctors can do their best work in industry.

At present, in Manchester and Edinburgh, a three months' course of general social and preventive medicine is given before six months of specialized industrial training. In other schools, however, much greater attention is given to general preventive medicine, and a shorter period of special industrial training. It may well be that the best solution lies somewhere between the two. For much industrial health work a sound clinical training is essential. The application of clinical knowledge to special industrial health problems rightly finds a place in this course, but doctors who take this course must already have a sound knowledge of clinical medicine.

Part-time Industrial Medical Officer.—The British Medical Association Report on the Training of the General Practitioner (1950), points out that over 3,000 general practitioners are already doing part-time work in industry. This is a sound arrangement and is of advantage to both doctor and industry. It enables the general practitioner to see for himself the manner in which many of his patients spend their working lives, while he benefits personally from contact with professional people other than those associated with medicine. It enables industry to secure the services of one who can often throw light on difficult human problems, as a result of his knowledge of social conditions outside the factory. The report draws attention to the need for providing training for these men. It would be quite unreasonable and unnecessary to expect them to possess a Diploma in Industrial Health, but a short introductory course of two weeks would do much to orientate the newcomer whose life had largely been spent in curative medicine. Experience during the war showed that such courses were of considerable value, and were well supported by general practitioners. Teaching in these courses must be imaginative and must largely depend upon discussion and practical demonstration. Such courses are likely to raise the quality of the work of all doctors practising in an industrial area, whether or not they hold a post in industry.

The Teacher and Research Worker.—At the present time some of the men most important to the development of this branch of medicine are those engaged in teaching and research, and the best training possible should be available for them. This should be wide and include a thorough training in some branch of medicine with, if possible, a higher qualification, followed perhaps by a fellowship or research post in one of the university departments of occupational health, followed again by a few years in industry, where as wide an experience as possible is secured. It is important that industry should recognize the need for such men and encourage their training.

Nurses

There are probably some 5,000 State Registered nurses in industry. Before considering the education required for a nurse, it is necessary to have a clear idea of her function. Is she a casualty dresser or has she a bigger role to play? I am convinced that she has, and that to use nurses in industry as nothing more than "finger tiers" is wasteful and wrong. The nurse in industry must be a health worker and she must be able to act for the doctor in many situations. She must, of course, have plenty of human understanding and commonsense, but this alone is not enough, she must also have a clinical sense. She must be a skilful and unobtrusive health educator. Though much can be done to prepare the nurse for her work in industry by giving her a post-graduate course, this will not secure its maximum results unless her standard of education and her basic training are good. There have been many critics of the present nurses' training and I do not wish to enter the fray, except to draw attention once more to the Working Party Report (1947), which emphasized that the explicit aim of nursing training should be the development of a nursing service in closer accord with modern ideas of social and preventive medicine. While, however, student nurses are required to staff our hospitals, and while so much domestic work is left for them to do, this aim is a long way off, but it is hoped it will not be lost sight of.

With the present shortage of nurses it is essential that the trained nurse who comes into industry should fill a position of responsibility. She must not be wasted. She should teach and supervise assistant nurses and industrial first aid attendants. This, in fact, should be one of her most important functions and she should be trained to teach.

Credit must be given to the Royal College of Nursing for the pioneer work it has carried out in this field over the last 16 years. During the war Birmingham University became the first university to undertake the training of industrial nurses. This experiment was successful and a flourishing school for industrial nurses now exists in that city. The Manchester University Department of Occupational Health has trained industrial nurses for three years. To meet the needs of many who have been unable to secure training a part-time course extending over two years has recently been started as an experiment. It is too early yet to pronounce on this scheme, but with proper safeguards it appears to have much to recommend it. The whole question of the best preparation of the nurse for industry is beset with difficulties. One thing only is certain, that we must on no account fail to use her services to the full.

There is much important work to be done which can safely and properly be delegated to assistant nurses and others. It is not my purpose here to discuss the matters relating to their supervision and training, but only to emphasize that they are members of the industrial health team, and their training is our responsibility.

One of the chief values to industry of medically trained personnel, be they doctors or nurses, is that they bring to industry a different outlook and a different ethic. This they do by virtue of their

basic training in our medical schools and hospitals, and though one has criticized this in detail, it cannot be too strongly held that the fundamentals of this medical education have been imparted in the traditional teaching of our profession.

Industrial Hygiene Engineer

No consideration of the training of the specialists needed for occupational health work in industry would be complete without reference to the industrial hygiene engineer. It is his duty not only to be able to appraise physical environmental conditions, but also to give technical help regarding any modifications and improvements required. Much of this work is carried out by factory inspectors, who have a wide and practical knowledge of these problems. The fact remains that industry needs more technical assistance than can be reasonably asked of a public servant.

In the U.S.A. considerable attention has been given to the industrial hygienist. In Harvard University the Department of Industrial Hygiene is under the able control of Professor Philip Drinker, an engineer. The basic training of these specialists may have been in one of several fields, engineering, chemistry, or physics, for example. Following upon this professional training and some practical experience in industry, a course extending over a year is given, and in Harvard this is taken side by side with the doctors.

There is little doubt that in this country we have lagged behind in this part of the field, and that steps should be taken to repair this omission. It would seem that those universities with departments of occupational health would make good training centres for these men, who could retain the full facilities and support of their own particular faculty in the university.

These specialists are important members of the industrial health team, and the need for training a few such men for British industry is urgent.

The responsibility for education in occupational health must rest with the universities. A university must be closely linked with the life of the community, and, in an industrial society such as ours, will find in occupational health a subject fully worthy of its close attention. It is right, therefore, that a few university departments of occupational health have been established. This has been made possible by the generosity of the Nuffield Foundation. The university department, however, can do little without the wholehearted support of industry, for both teaching and research in this subject require industrial communities as their laboratory. Industry will rightly demand that the responsible teachers of such departments will be fully aware of the pitfalls associated with fieldwork in an industrial community, but with such assurance should be prepared to offer full collaboration. In my experience this collaboration has usually been readily accorded.

The future development of industrial health services is at present the subject of enquiry by a Government committee. Should this committee recommend immediate expansion it would presumably be a Government responsibility to provide suitable training for personnel. On the other hand, should the committee decide that such action would be precipitate, it would presumably be left to industry to develop its own services. If these services are to make a real contribution to health and efficiency, the size of the problem must be appreciated, and industry would be well advised not only to go out to recruit its due share of the best brains in the medical profession to its service, but to pay due regard to their training. University departments of occupational health exist and are already doing important work; it is industry's responsibility to see they are used to the full, for only in this way will they be stimulated to adequate growth and be able to attract the right quality of recruits.

Recently the question of overlapping between the doctor and nurse in industry and in the National Health Service has been raised. There should be no such overlapping if the doctor and nurse know their proper functions. Industry has every right to a proper proportion of the medical personnel of the country, but it is responsible for seeing that they are not wasted. This means sound training for the special work they are to undertake.

I have tried to indicate that occupational health is concerned with the quality of life as it is affected by occupation, and is the joint concern and responsibility of industry, the educationist, and of medicine. Medicine's contribution is through research and service. At this early stage the growth and development of this branch of medicine must be a matter for experiment. For this, inspired and informed leadership is essential, and much of this must come from the ranks of our profession—particularly from those who work in industry. This applies not only to those who have the actual task of teaching, but to those who are working in the field. The future development of this branch of medicine depends in no small measure on the educational opportunities we provide for it, and these must be related to demand. Talk about education and industrial health is of little value unless we have a clear idea of what we are after. This, I repeat, is the responsibility of each of us and not just the few. Let each individual define

his aims and objects and those he considers to be proper to this branch of medicine, for what Montaigne said 400 years ago is abundantly true today :

"No wind makes for him that hath no intended port to sail unto."

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ENVIRONMENTAL CONDITIONS IN THE MERCURY MINE OF IDRIA*

BY

V. B. VOUK, M. FUGAŠ, and Z. TOPOLNIK

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The history of mercury mining in Idria has been excellently described by Teleky (1912) and Kropač (1912). Some historical data on mercury poisoning in Idria is also given by Rosen (1943). From these works the data for the following short historical introduction have been taken.

Mercury deposits in Idria have been known since 1490 or 1497. It is said that mercury in Idria was discovered by a cooper, who found mercury droplets when filling his container from a nearby well. He founded a mining company which was very soon taken over by the local lord. After 1580 the mines became the property of the prince of the province and they were left under the management of the Austro-Hungarian state till 1918, when the Idria district became part of Italy. After the second world war the Idria mines were ceded to Yugoslavia.

Mining was at first in the bituminous shales containing free mercury, but very soon the exploiters turned to cinnabar deposits. The smelting process was very primitive at that time : alternating layers of ore and wood were covered by earth and set alight. The construction of aludel-furnaces, first used in Spain, was an important technological development. In 1785 the Austrian government concluded a trade agreement with Spain and thus undertook to deliver large quantities of metallic mercury. The first measure designed to increase the output of the Idria mines was a regulation providing for the mines and the smelting plant to work all the year through. Before this the smelting plant worked only during three winter months. At the same time the smelting process was improved by the construction of vertical flame furnaces by Leithner in 1787. In 1803 a big fire broke out in the mines and a large part of the pits was flooded. In 1842 the first furnace operating continuously was built in Idria.

Mercury poisoning among Idria miners was described at an early date, one of the first authors of such descriptions being Paracelsus (1553). Teleky (1912) quotes papers by Pope (1665), Fallopius (1665), Ramazzini (1700), Keyssler (1740), Scopoli (1761 and 1789), Hacquet (1766 to 1773), Hermann (1857) and many others .A very interesting description of mercurialism in Idria comes from Andrea Mattioli in the sixteenth century (Rosen, 1943 ; Glesinger, 1950). But by far the best and most exhaustive study of this problem is the monograph by Teleky (1912). The problem in the period 1946 to 1950 has been recently described by Hribernik (1950).

Although mercurialism in Idria was very often described from the purely medical point of view, studies of environmental hygiene in the mine and the smelting plant are scarce. Hacquet, a surgeon in Idria from 1766 to 1779, proposed that miners should take hot baths after work and drink milk as preventive measures against mercury poisoning. General body cleanliness, special clothes for work, and the frequent exchange of workers at particularly dangerous working places were measures often recommended by other earlier writers. As far as we know, there was no attempt yet to make a quantitative study of health hazards in the mine and the smelting plant of Idria. The available literature does not offer any data concerning the determination of mercury concentration in the working atmosphere of any other European mercury mine. We do not know whether such air analyses have been performed in the United States, since the report by Davenport and Harrington (1941) was not available.

Technology and Hygienic Problems

In Idria, mercury occurs as cinnabar (HgS) and as native quicksilver. The average content of mercury in the ore is 0·7%. The cinnabar-bearing rock is dolomite, but sometimes also the sandstones of the Wengener group. Native mercury is found in places where dolomite is found in contact with the Geiltal bituminous shales, as well as in the

*A paper read to the Conference on Industrial Hygiene in Zagreb in July, 1950.

FIG. 1.—Winning of native mercury

FIG. 2.—Cinnabar in dolomite

[*Facing page* 168

TABLE I. RESU[illegible] INVESTIGA[illegible]

1 No.	2 Date	3 Location	4 M.P. Character	5 Operation	6 Ventila-tion	7 Personal Protection	8 t_d (°C.)	9 t_w (°C.)	10 R.H. (%)	11 w (m/sec)
1	19.5.50	III.L. 9 G.		Hand picking	+	—	25·0	21·0	73	0·11
2	17.5.50	III.L. 10 G.		Picking (not in work)	+	—	24·0	22·0	84	0·16
3	17.5.50	III.L. 10 G.		Picking (not in work)	—	—	25·0	23·0	84	0·10
4	16.5.50	I.L. 8 G.		Picking (not in work)	—	—	23·5	22·0	88	0·07
5	16.5.50	I.L. 9 G. W.P. 1		Picking (not in work)	—	—	21·5	20·0	87	0·08
6	26.5.50	I.L. 9 G. W.P. 2		Hand picking	+	—	22·5	21·0	92	0·14
7	18.5.50	IV.L. W.P.1		After work	+	—	19·0	17·5	86	0·07
8	18.5.50	IV.L. W.P.2		After work	—	—	19·5	17·5	86	0·13
9	22.5.50	IV.L. W.P.3		Hand picking	—	—	21·0	18·5	91	0·16
10	23.5.50	III.L. 9 G. W.P.1		Drilling	+	Respirator M.S.A.	25·0	23·0	92	0·10
11	23.5.50	III.L. 9 G. W.P.2		Hand picking	—	—	23·0	22·0	92	0·18
12	20.5.50	VI.L. 4 G. W.P.1		Hand picking	—	—	23·0	21·5	88	0·10
13	22.5.50	VI.L. 10 G.		Drilling	—	Respirator M.S.A.	18·5	17·5	91	0·11
14	20.5.50	VI.L. 4 G. W.P.2		—	—	—	22·5	21·5	9[illegible]	0·10

native mercury in shale.

cinnabar in dolomite.

native mercury in shale + cinnabar in dolomite.

cinnabar in dolomite + gangue.

RESU... INVESTIGATIONS IN PIT I

10 R.H. (%)	11 w (m/sec)	12 E.T. (°C.)	13 τ (min.)	14 C_{Hg} (mg./m³)	15 C_{max} (mg./m³)	16 $\frac{C_{Hg}}{C_{max}} \cdot 100$	17 $\frac{C_{Hg}}{Co}$	18 Remarks
73	0·11	22·5	420	2·00	19·7	10·2	20·0	..
84	0·16	21·9	120	3·20	18·1	17·7	32·0	..
84	0·10	22·8	120	5·90	19·7	30·0	59·0	..
88	0·07	22·3	90	4·50	16·5	27·3	45·0	..
87	0·08	22·0	90	1·20	14·0	8·6	12·0	..
92	0·14	20·8	320 240	1·65 1·20	16·0	10·3 7·5	16·5 12·0	Selenium sulphide C_{Hg} : 1·15 mg/m.
86	0·07	17·9	120	1·45	12·2	11·9	14·5	..
86	0·13	17·0	120	2·30	12·3	18·7	23·0	..
91	0·16	18·8	120	1·10	14·0	7·9	11·0	..
92	0·10	23·4	30	0·19	19·7	1·0	1·9	With filter
			30	1·10		5·6	11·0	Without filter
92	0·18	21·5	180	0·43	16·5	2·6	4·3	..
88	0·10	21·7	120	0·08	16·5	0·5	0·8	..
91	0·11	17·4	60	0·05	11·2	0·5	0·5	With filter
			60	0·87		7·8	8·7	Without filter
9..	0·10	21·5	120	0·21	16·0	1·3	2·1	..

The following abbreviations are used in the tables: L = level, G = gallery (sub-level), W.P. = working place, M.P. character = mineralogical and petrographical characteristics of the rock face, t_d = dry bulb temperature, t_w = wet bulb temperature, R.H. = relative humidity, w = velocity of air movement, E.T. = effective temperature (basic scale), τ = sampling period, C_{Hg} = mercury concentration in air, C_{max} = saturation concentration of Hg-vapour at the corresponding dry bulb temperature. (Data extracted from tables on mercury vapour pressure, Hodgman, 1946.) Co = maximum allowable concentration of mercury in air, i.e., 0·1 mg./m³ (American Standard, 1943).

TABLE 2. RESU

1 No.	2 Date	3 Location	4 Operation	5 Ventilation	6 Personal protection	7 td (°C.)	8 tw (°C.)
1	18.5.50	Shaft furnace I, II, on the top	Charging	Natural	..	20·5	15·0
2	14.10.49	Shaft furnace I, II, on the top	Charging	Natural	..	13·0	10·0
3	19.5.50	Shaft furnace III, IV, on the top	Charging	Natural	..	18·0	14·5
4	22.5.50	Špirek furnace III, on the top	Charging	Natural	..	29·0	20·5
5	14.10.49	Špirek furnace III, on the top	Charging	Natural	..	15·0	11·5
6	23.5.50	Špirek furnace IV, on the top	Charging	Natural	..	27·0	18·0
7	14.10.49	Špirek furnace IV, on the top	Charging	Natural	..	18·0	12·5
8	24.5.50	Under the Špirek furnace	Discharging	Natural	..	24·5	17·5
9	14.10.49	Under the Špirek furnace	Discharging	Natural	..	19·5	14·0
10	23.5.50	Špirek furnace VI	Charging	Natural	..	28·5	19·0
11	17.5.50	Soot-press	Exeli press Working	Natural	..	18·0	12·0
12	15.10.49	Soot-press	Exeli press Working	Natural	..	..	..

VESTIGATIONS IN THE SMELTING PLANT

9	10	11	12	13 Conc.	14 Hg	15
R.H. (%)	w (m/sec.)	E.T. (°C.)	τ (min.)	D (mg./m³)	Se (mg./m³)	Remarks
54	..	..	480	0·26	0·10	Charging every 3 hours. Continuous sample
69	..	..	30	0·88	..	Sample taken only during charging
69	..	..	480	1·10	0·12	Shaft furnaces are filled with cinnabar ore
46	..	..	360	0·24	0·15	Continuous sample
66	..	..	30	0·33	..	Sample taken only during charging
41	..	..	240	0·88	0·37	Continuous sample
53	..	..	30	0·23	..	Sample taken only during charging
40	0·80	..	480	0·43	0·08	Continuous sample
54	0·10	..	30	0·50	..	Sample taken only during charging
39	0·45	..	240	0·17	0·20	Charging with rich ore (native mercury)
49	0·14	15·5	240	0·82	0·58	..
..	..	..	30	0·68	..	..

TABLE 3

RESULTS OF INVESTIGATIONS IN PIT II

No.	Date	Location	M.P. Character	Operation	Ventilation	Personal Protection	t_d (°C.)	t_w (°C.)	R.H. (%)	w (m/sec.)	E.T. (°C.)	τ (min.)	C_{Hg} (mg./m³)	Remarks
1	24.5.50	IX.L. 3 G.		Transport of ore	..	..	17·5	17·0	95	0·17	14·9	120	0·05	..
2	24.5.50	IX.L. 3 G.		Picking (not in work)	..	..	15·5	15·0	95	0·15	13·8	120	0·05	..
3	25.5.50	XII.L. 5 G.		Transport of ore	..	..	..	..	..	..	..	120	0·08	..
4	26.5.50	XII.L. 9 G.		Picking and transport	..	..	20·0	19·0	91	0·25	17·7	120	0·50	30 min. Drilling
5	25.5.50	XII.L.		Drilling	..	Respirator M.S.A.	19·0	17·5	96	0·16	17·0	60	0·10	With filter
												60	0·24	Without filter
6	26.5.50	XIII.L. 12 G.		Transport of ore	..	..	20·0	19·0	91	0·15	18·5	120	0·14	Dust ?

From the point of view of the industrial hygienist the working places in Idria may be divided into three groups: (1) Pit I, where only cinnabar is won, and where no poisoning hazard exists; (2) Pit II, where free mercury occurs beside the cinnabar, and the danger is obvious; and (3) the smelting plant in which especially dangerous operations are the charging of shaft-furnaces, processing of "soot", filling of steel bottles, the monthly cleaning of condensation pipes, and the periodic cleaning of the chimney.

In order to show the scope of health hazards in Idria, we quote some data on mercurialism in Idria from 1946 to 1950 (Hribernik, 1950). In 1946 there were 81 cases of poisoning (seven severe). In 1947 the number of workers poisoned by mercury fell to 27 but in 1948 this figure was again higher and reached 51. In 1949 Hribernik reported 93 cases, and in the first half of 1950 there were 53 poisonings. All these cases were subacute, chronic, or chronic-stationary poisonings.

Evaluation of Hazards

The Institute of Industrial Hygiene of the Yugoslav Academy of Sciences and Arts in Zagreb undertook to investigate the possibilities of making the working environment in the Idria mines less dangerous to the miners' health. The first part of this programme was the determination of the average concentration of mercury in the air breathed by the miners and the smelting plant workers, and the measurement of thermal environment. These investigations were carried out in two parts, in October, 1949, and in May, 1950. The measurements relate to 28 working places, and 43 air samples were taken for the determination of atmospheric mercury.

Analytical Methods

Thermal Environment.—Dry and wet bulb temperatures were measured by means of a sling hygrometer. The air velocity was recorded with a heated-thermometer anemometer (Yaglou) and a kata-thermometer (38–35°C). The relative humidity and the effective temperatures were then calculated. The effective temperature was evaluated by means of a nomogram (Bedford, 1946) calculated against the basic effective temperature scale, as most miners work stripped to the waist.

Determination of Mercury.—Mercury may enter the body by inhalation and by absorption through the skin and the mucous membranes. It is, however, the atmospheric mercury that causes most trouble. Mercury in air may occur as mercury vapour, as mercury aerosol, or as mineral dust contaminated with metallic mercury. All three forms of atmospheric mercury present a health hazard. Therefore, we had to apply an analytical method by which it was possible to estimate the total mercury content in the working atmosphere. We used the colorimetric dithizone method as described by Milton and Duffield (1947) with slight modifications.

The samples were taken by aspirating air through two midget impingers connected in series and filled with 5 ml. absorbing solution (4 ml. of bromine in 100 ml. of 8% NaOH) each. Glass beads were put in the impingers in order to increase the absorption surface and to reduce the foaming. The air was aspirated through the impingers at a speed ranging from 0·5 to 1·6 litre per minute by means of hand and electrically driven pumps. The sampling period varied from 30 minutes to eight hours. The size of samples ranged from 30 to 580 litres of air. (The size of each sample was checked separately by means of a precision wet-meter and the manometer attached to the pump.)

The sampling period in the pits was usually two hours. The procedure was as follows:

The air was drawn through the impingers for 30 minutes, then followed an interval of about one hour. This was repeated four times within the working shift of eight hours. In places where much dust was produced (for example, in drilling and exploration work) a fibrous filter ("Whatman" filter paper No. 42) was attached to impingers in order to prevent the dust from entering the absorption liquid. In the smelting plant continuous samples of four to eight hours were taken.

All colorimetric measurements were performed with a Beckman spectro-photometer at the wavelength of 450 mμ.

In some working places, where electricity was available, the concentration of mercury vapour was estimated by means of a "General Electric" selenium sulphide mercury vapour detector. This indicator is sensitive to mercury vapour only, but not to mercury aerosols. The advantage of this method is that selenium sulphide paper does not react with cinnabar dust.

Results.—The results of measurements are given in Tables 1—3.

The data have been calculated assuming that all atmospheric mercury exists as mercury vapour, which is not strictly correct. However, "saturation" as defined in the heading of column 16, Table 1, is a very useful parameter showing the overall contamination of air with mercury. Mercury vapour concentrations, determined by means of selenium sulphide paper, are noted in the column "Remarks". A special note is put also for cases where a fibrous filter was attached to the sampling apparatus.

Table 2 refers to the smelting plant. Here the concentrations of mercury vapour, obtained by selenium sulphide indicator, are entered in a separate column (Se). Those obtained by the dithizone method are marked by D. The air movement in the smelting plant was also measured, but it changed so rapidly that the results could not be given by a single number, and have therefore been omitted.

Discussion

As shown in the tables, the dry bulb temperature in the Pit I ranged from 18·5 to 25°C, in Pit II from 15·5 to 20°C, and in the smelting plant from 18 to 29°C. Considering that these figures refer respec-

tively to underground working places and to work near roasting furnaces, they are rather low. Data on relative humidity are not so satisfactory. Relative humidities of 73–92% found in Pit I, and of 91–96% in Pit II are very high. The air velocity in Pit I (0·07–0·18 m/sec.) and in Pit II (0·15–0·25 m/sec.) is inadequate owing to the lack of mechanical ventilation which is found in a few places only.

The values of effective temperature range from 17–23·4°C (Pit I) and from 13·8–18·5 (Pit II) showing that overall climatic conditions in Pit II are considerably better than in the Pit I. However, the effective temperature does not give an adequate picture of climatic conditions in the pits. Thus in many working places in Pit I we experienced a feeling of discomfort which may be described as " too hot and stuffy ", although the effective temperature was not particularly high. This was probably due to the fact that the air movement was very low and showed no variability.

Mercury concentrations found are rather high and amount to 0·05–5·9 mg/m^3 (Pit I), 0·05–0·50 (Pit II), and 0·17–1·1 mg/m^3 (smelting plant). An objection may be raised that these concentrations are in general too high owing to the contamination of samples with cinnabar dust. We had in mind this possibility and we tried to eliminate it as much as possible. Whenever it seemed that contamination with cinnabar was likely to occur, we put a note in the table. It is natural that the values of mercury concentrations obtained by means of selenium sulphide indicator are lower than those obtained by the dithizone method, but it does not necessarily mean that dithizone samples were contaminated by cinnabar, as we have to bear in mind that the selenium sulphide does not react with mercury aerosols. In general the results agree well with medical experience concerning the relative danger at different working places. The results obtained in October, 1949, fully correspond to those obtained in May, 1950.

General hygienic conditions in the Idria mines are satisfactory. The washing and toilet facilities are adequate. Hot showers are always available after work. There are also good wardrobes where miners change their working clothes before leaving the plant. Those working at dangerous points receive an extra ration of bacon. There is a part-time medical officer, who examines twice a month all those working at danger points. On the basis of these periodical examinations, those workers who show any symptoms of mercury poisoning are immediately withdrawn, or given other jobs presenting no mercury hazard. Further medical control consists in the frequent alternation of workers at exposed places. Thus in Pit I, Level I, the miners are changed every four days or sooner if necessary. All work in a mercury-contaminated atmosphere is carefully recorded. Workers performing dusty operations wear dust respirators (usually of Mines Safety Appliances type). At present there are, however, no respirators against mercury vapour in use.

Although the information obtained so far from our investigations is incomplete, it nevertheless suggests some further protective measures, such as (1) the improvement of ventilation (Pit I), which would reduce the concentration of mercury in air to a certain extent, and at the same time make thermal conditions better ; (2) the use of respirators against atmospheric mercury (in such operations as winning native mercury, charging furnaces, and cleaning the main chimney) ; (3) the use of hose-masks for cleaning of condensers ; and (4) wet methods in drilling.

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LEAD ENCEPHALOPATHY IN A COOPERAGE

BY

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Advances made in the industrial field have led not only to a reduction in the total number of cases of plumbism, but also to a relative decrease in the severe forms of the disease, including encephalopathy. Of 6,638 cases of plumbism occurring during the years 1900 to 1909, 262 were cases of encephalopathy, an incidence of 3·9% (Legge, 1934), whereas of 634 cases notified to the Factory Department during the years 1939 to 1948, only five showed symptoms of encephalopathy, less than 0·8%. It is generally agreed that severe exposure by inhalation of dust, fume, or vapour is necessary to produce encephalopathy. Today, in large industrial concerns, inspection and control are so thorough that such exposure seldom arises. The cooperage described in this paper had escaped inspection, and two coopers contracted lead poisoning, one of whom developed encephalopathy, before the presence of a severe lead hazard was brought to light.

Of all the manifestations of inorganic lead poisoning in the adult, encephalopathy is the most serious and the least common. The last cases to be recorded in Britain would appear to be those of Chalmers (1940) and Nussey and Drybrough-Smith (1940). Encephalopathy in children differs somewhat from that seen in adults, the whole clinical and pathological picture suggesting a more intense reaction : it will not be discussed.

Grisolle (1836) distinguished three main forms of encephalopathy : convulsive, comatose, and delirious, of which the convulsive was the most serious and the most common. As Tanquerel (1839) pointed out, these forms occur more often in combination than singly. The onset of encephalopathy may be sudden. When apparently well, the patient may fall in an epileptiform attack or pass rapidly into coma or mania. More often there is warning, either in the form of other manifestations of lead poisoning or in the occurrence of various symptoms which frequently precede the onset of frank encephalopathy, namely intense headache; restlessness ; depression ; insomnia and disturbed dreams ; hallucinations ; pallor ; faintness ; vertigo ; disturbances of vision ; and transient paralysis and aphasia, paræsthesiæ, and tremors of the hands and face (Aub, Fairhall, Minot, and Reznikoff, 1925 ; Hamilton, 1925).

The disturbance of cerebral function caused by lead varies in degree, from the restlessness, irritability, impairment of memory and lack of concentration encountered in all forms of lead poisoning, to the confusion, delirium, mania, and coma of advanced encephalopathy. The delirium does not appear to differ essentially from severe delirium produced by other causes, but the concurrence of convulsions may present a characteristic picture. These may take the form of major epilepsy with loss of consciousness, but Tanquerel (1839) considered that partial preservation of consciousness was more common.

Papillœdema, retinal hæmorrhages, and subsequent optic atrophy are well recognized complications of lead encephalopathy. Primary optic atrophy due to lead is also said to occur. In the potteries, Prendergast (1910) found total blindness in 7·7% and partial blindness in 10·2% of women with plumbism. Although the onset may be of dramatic suddenness, it is usually gradual, and eventual recovery is the rule. Transient attacks of loss of vision, affecting the whole or part of the visual field, have also been described, and are usually attributed to spasm of the retinal or cerebral arteries. Paralysis of the extrinsic eye muscles is not uncommon ; abnormalities in pupillary reactions and accommodation are rare. At one time or another lesions of all the cranial nerves have been attributed to lead. Arterial hypertension not infrequently accompanies encephalopathy, and there may be some degree of pyrexia. Some impairment of mentality, either temporary or lasting, was found by Alice Hamilton (1925) in 19 out of 59 cases of encephalopathy reviewed by her. One of her cases suffered from recurrent epilepsy after apparent recovery and similar cases have been described by other writers. As might be expected,

an anxiety state and hysteria may follow encephalopathy in some cases.

The relationship between lead on the one hand and arteriosclerosis, nephritis and hypertension on the other, is now a matter of dispute, but in the past lead was accepted as a causal factor in these conditions, and cases of cerebral arteriosclerosis and of uræmia have been included in accounts of saturnine encephalopathy. The mortality rate in different recorded series varies from 25% to 75% (Aub and others, 1925). Hamilton (1925) and Cantarow and Trumper (1944) have reviewed the literature of this subject.

Many conflicting reports of the pathological changes in encephalopathy have been published. Aub and his co-workers (1925) concluded that the condition was primarily meningitis with secondary involvement of the brain, " a meningo-encephalopathy ". The more recent work of Freifeld, Tuthill, Staemler, Winkelman, and Eckel and Rhea (quoted by Blackman, 1937) support the older view that lesions in the nervous system are secondary to changes in the vessels. Blackman (1937), who made detailed pathological studies of 22 children who had died of encephalopathy, concluded that " the lesions in the brain are for the most part dependent on vascular damage and the collection of exudate ".

The most frequent finding in the cerebrospinal fluid is a raised pressure. An increase in protein and in the number of lymphocytes may also occur, but these changes are seen more often in children than in adults, in whom they do not appear to be a constant or marked feature. Mosny and Malloizel (1907), quoted by Aub and others (1925), found that the cell count averaged 100 lymphocytes per c.mm. in encephalopathy, but their findings have not been borne out by subsequent observation.

Cantarow and Trumper (1944) observed concentrations of lead ranging from 0·001 to 0·040 mg. per 100 ml. of cerebrospinal fluid in patients with no history or evidence of abnormal exposure to lead, and concentrations as high as 0·35 mg. per 100 ml. in subjects with lead poisoning with no nervous manifestations. They quote Duensing as reporting values of 0·22 mg. and 0·493 mg. per 100 ml. in two cases of lead poisoning with neurological manifestations.

Since 1923, when 149 cases of encephalopathy due to tetra-ethyl lead occurred in the United States of America, attention has been focused on this form of lead poisoning, and series of cases have been reported by Kehoe (1925), Machle (1935), Cassells and Dodds (1946), Singh (1949), and others. The fat soluble nature of tetra-ethyl lead predisposes its early localization in tissues of high fat content, in particular the nervous system (Kehoe, 1925). Its action on nervous tissue is probably therefore direct and not, as appears to be the case in inorganic lead poisoning, indirect through changes in the vessels. The cerebral symptoms of tetra-ethyl lead poisoning differ in many respects from those of inorganic lead encephalopathy. Convulsions are rare, papillœdema, optic atrophy, loss of vision, temporary pareses, paræsthesiæ and aphasia do not apparently occur, and no permanent cerebral sequelæ were noted in over 80 cases (American Public Health Association, 1943). The pressure of the cerebrospinal fluid may be raised during delirium, but shows no evidence of a meningeal reaction (Machle, 1935). The marked drop in blood pressure and slowing of the pulse so frequently seen in tetra-ethyl lead poisoning are not features of inorganic lead encephalopathy. For these reasons it would be unwise to draw any conclusions about inorganic lead encephalopathy from the findings in tetra-ethyl lead poisoning.

Case Report

The patient, a man of 39 years, was first seen as an out-patient on March 30, 1949. His main complaints were of headache, vomiting, and general weakness, but sugar and acetone were found in his urine and on questioning he admitted to loss of weight and polyuria. A provisional diagnosis of diabetes mellitus was made.

On admission to hospital the next day there was no acetone and only a trace of sugar in his urine, and it was clear that diabetes was not the cause of his symptoms. He was somewhat confused and his headache was extremely severe. He volunteered no information and tolerated questioning and examination with reluctance. No suspicion of a lead hazard was aroused at this time, and much of the following history was obtained at a later date when the patient was more coherent. He had been a cooper for over 20 years but for eight months had been working for a firm chiefly concerned with the repair of barrels used for white lead. One of his mates had been off work with colic and constipation. He was a very heavy beer and spirits drinker, although he had been taking less during the last week or two. His father, also an alcoholic, had died of cirrhosis of the liver. Except for a chronic winter cough he had previously been healthy.

For six months he had been troubled by headache, general weakness and morning anorexia, and had lost about a stone in weight during that period. The headache had been at first slight and infrequent, but recently it had been practically continuous and of such severity that the patient could not remain still during the day and had slept little at night. It was mainly frontal, was aggravated by stooping, occurred at no particular time of the day, and for six weeks had been associated with vomiting. Although he had first noticed a general weakness and ease of fatigue, latterly this had been most marked in his right arm, the grip being particularly

affected so that he had found it difficult to use his hammer at work. At the onset of his symptoms he had suffered from obstinate constipation lasting two weeks and associated with lower abdominal colic. There had been no further constipation, but occasional bouts of diarrhœa.

For six weeks he had suffered from periodic diplopia and blurring of vision, slight deafness and occasional tinnitus in both ears. He had also experienced attacks of dizziness when walking, which caused him to stagger, though he had not fallen; there was no actual sense of rotation and he did not deviate in any particular direction. On one occasion there had been transient weakness of both hands, lasting only a few minutes, and once whilst at work he had become suddenly but temporarily blind. It was not clear whether this was a total or partial visual field defect. He could only remember that for a minute or two he had been unable to see his mate, who had dismissed the matter with some cryptic remark about his drinking habits. He had a slight cough, and a few days before admission had coughed up about two ounces of bright red blood. He gave a history of polyuria for some months and of recent thirst, and he had noticed an unpleasant taste in his mouth which might be described as metallic. About ten days before admission his doctor had given him a cough mixture; his symptoms had been very severe during the following week. (This prescription was dated March 19, 1949, and contained 2½ gr. potassium iodide in each dose. It is thought that he took very little of this medicine, and the exacerbation of symptoms at that time may have been coincidental.)

Examination on Admission.—The patient was of average muscular development, with little subcutaneous fat. His eyes were watery and suffused, his skin and mucous membranes pale, and there was slight but definite beaking of the finger nails. The pulse, temperature, and respirations were normal. The breath was offensive and the mouth foul, with furred tongue and gingivitis. The blood pressure was 125/85 mm. Hg, and no abnormality was detected in the heart, lungs, abdomen, or external genitalia. Rectal examination was normal.

The findings in the nervous system were: fine tremor of the lower lip and outstretched hands; speech normal, but cerebration slow and memory poor; no evidence of meningism; pupils normal in appearance and reactions; bilateral slight papillœdema, more marked on the right; retinal vessels normal with no hæmorrhages or exudates; slight weakness of the right external rectus muscle, but no diplopia during examination and no nystagmus; slight middle ear deafness on the left side; other cranial nerves intact; slight generalized muscular weakness; loss of power most marked in the right arm, the grip of the right hand being particularly affected; no wasting or fibrillation; no spasticity; tendon reflexes brisk and equal; superficial reflexes normal; all forms of sensation normal and Romberg's sign absent; no intention tremor or dysdiadochokinesia, but movements of the arms and legs shaky and the gait unsteady.

At this stage the suggested diagnosis was bronchial carcinoma with secondary deposits in the brain. Radiographs of the skull and chest were, however, normal. Lumbar puncture gave a clear fluid under very high pressure, some 10 ml. of which was cautiously withdrawn. Examination of the fluid revealed protein 60 mg. % with a slight increase in globulin; gold curve 0112210000; sugar 70 mg. %; chlorides 650 mg. %; no increase in cells: Wassermann reaction negative in all dilutions. The blood Wassermann reaction was also negative, the non-protein nitrogen 35 mg. %, and the urine contained no albumin, casts, or red cells. An electrocardiogram was normal.

Progress.—The patient was given magnesium sulphate by mouth, and, when his condition permitted, hypertonic retention enemata. Severe headache and frequent vomiting persisted, although temporarily abolished by lumbar puncture. He became incontinent of urine and fæces. Papillœdema increased, and a small retinal hæmorrhage appeared in the right eye. He was much troubled by diplopia. During the day he was more confused, and was difficult to control. At night he passed into a noisy, agitated delirium, shouting, swearing, and dancing about on his bed. When not thus engaged he maintained a ceaseless rambling chatter. He was disorientated as to time and place, and clearly the victim of frightening hallucinations. Repeated injections of soluble phenobarbitone were necessary to control the delirium.

Although there was amnesia for the nights on which he had been delirious, he was able, some days later, to give an account of the hallucinations from which he had suffered at other times. They were mostly visual and had first occurred some days before admission, when he was much troubled by a man in woman's clothing, who came into the bedroom at night, though the door was locked, and attempted to get into the bed. When the patient tried to come to grips with him he disappeared. More recently, while in hospital, the hallucinations had been of a terrifying nature; enormous black men thrusting at him with their spears or men with machine-guns trained on him. Sometimes there was micropsia, in which all the doctors had tiny hands. To a lesser extent he suffered from auditory hallucinations, people calling him by name and passing offensive remarks about him.

On several occasions his urine contained a trace of sugar, but as the blood sugar remained normal this glycosuria was presumably due to a low renal threshold associated with the raised intracranial pressure.

By the fifth day a slight improvement in his condition was apparent. Although noisy and confused at night, he was more easily restrained, and by day he appeared rational. Headache remained severe. During the course of a further examination at this time a blue line was observed near the gum margin, most marked where infection was severe, and myoidema was elicited in the muscles of the shoulder girdle and upper arms. For the first time lead poisoning was suspected. The possibility of a lead hazard was admitted by the patient and confirmed by a visit to the cooperage where he had worked.

Examination of the blood revealed marked punctate basophilia with increased polychromasia; hæmoglobin 9·7 g. (65% Haldane); red blood cells 3,500,000; colour index 0·9; reticulocytes 4·5%; white blood cells 9,500, with a normal differential count. A red cell fragility test showed marked increased resistance to hypotonic salines. The Van den Bergh test was negative and the icteric index 2 units; the thymol turbidity 2 units and the alkaline phosphatase 5 units. So far as these tests were concerned there was no evidence of liver damage. Urea concentration and clearance tests gave results within normal limits.

Lumbar puncture was repeated on the sixth day, about 20 ml. of cerebrospinal fluid being withdrawn. The pressure was still extremely high, and a rise of cell count to 10 per c.mm. was noted. Analysis of blood and cerebrospinal fluid revealed a high lead content: blood 0·33 mg. per 100 g.*, cerebrospinal fluid 0·06 mg. per 100 g. The patient was then given calcium lævulinate intravenously (20 ml. of a 10% solution), followed by a high calcium and phosphorus diet, supplemented by calcium lactate orally (5 gr. t.d.s.). No extra iron or vitamins were administered. Sedatives at night and magnesium sulphate by mouth and rectum were continued. Complete relief of headache following lumbar puncture lasted some eight hours on this occasion. Urine collected on the seventh day contained 0·5 mg. of lead per litre*, and the total urinary excretion of lead during the 24 hours was 0·9 mg.

From this time the patient's condition improved more rapidly. Headache occurred less frequently and was easily relieved by mild analgesics. His behaviour was occasionally strange at night, but in general he slept well and assured us with obvious relief that there were "no more devils". Incontinence ceased. His appetite improved and he lost no more weight, although it was several weeks before he began to gain. Muscular power increased. The retinal hæmorrhage was absorbed and the papillœdema regressed. Clinical improvement was accompanied by a fall in the pressure of the cerebrospinal fluid. On the tenth day this was 150 mm. on the thirteenth day 95 mm. and on the fifteenth day 80 mm. of cerebrospinal fluid. The blood pressure remained normal.

This satisfactory progress was interrupted on the nineteenth day by a noticeable recurrence of symptoms. The patient felt "off colour", and complained that for a short time he had been unable to control the movements of his hands. Later severe headache and insomnia returned. On the twenty-second day the pressure of the cerebrospinal fluid was considerably increased, although the protein had fallen to 50 mg.% and the cells to 2 per c.mm. By the twenty-fourth day he was confused, irrational and emotionally unstable, weeping at the slightest provocation. There were no further hallucinations, but he suffered from various delusions, accusing the nursing staff of keeping him awake at night, and of leaving clues around the ward for him to solve, so that he might work out his own salvation. This setback was short-lived. By the twenty-seventh day he was mentally normal and there was no headache; the cerebrospinal fluid pressure was 250 mm. The blood pyruvic acid was 0·8 mg.% on the twentieth day and 0·95 mg. % on the twenty-sixth day. With the method employed for estimation both these figures are within normal limits.

On the thirtieth day there was still slight papillœdema in the right eye, and some weakness of the right arm. There was no tremor, ataxia, or incoordination. By the thirty-seventh day the only abnormalities on physical examination were a lead line, myoidema of the arms, and clubbing of the fingers. The cerebrospinal fluid was normal. The patient felt well. He was then given a normal diet without supplementary milk or calcium. For a few days there was a return of headache, and after ten days a rise in the cerebrospinal fluid pressure to 200 mm. was noted. For the next two weeks a low calcium diet was given. There was no return of headache. More drastic methods of de-leading were not attempted.

The blood picture had meanwhile shown a steady improvement. The number of stippled cells per million red cells fell from 16,000 on the fourteenth day to 4,500 on the twenty-second day. Dr. Charles St. Hill reported on Giemsa stained marrow films obtained on the twenty-sixth day:

"There is considerable increase in red cell production. The immature cells appear normal up to the late (polychromasic) normoblast stage. At this stage about 4% of normoblasts show punctate basophilia. The mature red cells from the marrow contain 23,000 stippled cells per million red blood cells." These findings are in agreement with the recent work of McFadzean and Davis (1949). We were, however, unable to obtain a positive reaction for iron in the stippled cells, using films fixed in methyl alcohol. A red cell fragility test on the thirty-seventh day was normal. Examination of blood on the sixty-second day showed hæmoglobin 11·8 g. (80% Haldane); red cells 4,500,000; reticulocytes 2%; stippled cells 700 per million red blood cells.

The progressive decrease in the concentration of lead in the blood and cerebrospinal fluid and in the amount excreted daily in the urine can be seen in Table 1 and Fig. 1. This fall was maintained during relapse, and was not apparently affected by reduction of the calcium intake.

After nine weeks in hospital the patient was discharged. He had no complaints. The lead line was less distinct; myoidema and finger clubbing were still present. The cerebrospinal fluid was normal; the pressure 120 mm. Shortly afterwards he obtained light work with another firm of coopers where there was no exposure to lead.

Six months after his first admission to hospital the patient's only complaint was an occasional feeling of faintness, chiefly when he was worried; otherwise he claimed to be in perfect health. For the previous two months he had been taking a normal diet without extra milk and had limited himself to two pints of beer a day. His wife, however, complained that he was now extremely irritable and hypersensitive and made

* In normal persons the concentration of lead in the blood ranges from 0·01 to 0·06 mg. per 100 g. of whole blood, and the concentration of lead in the urine ranges from 0·01 to 0·08 mg. per litre of urine in samples of 1 litre or more (American Public Health Association 1943).

constant accusations against her of infidelity for which there was apparently no justification. She confirmed that he was now sober in his habits.

Physical examination revealed no abnormality. The lead line had completely disappeared from his gums, and, surprisingly, the finger nails were no longer beaked. Blood pressure was 140/85 mm. Hg; cerebrospinal fluid normal; blood count: hæmoglobin 14·8 g. % (100% Haldane); red blood cells, 5,100,000; no stippled cells seen; non-protein nitrogen 35 mg. %; no abnormality detected in the urine. The daily urinary excretion of lead had diminished, but the concentration of lead in the blood had risen slightly since the last estimation (Table 1).

When next examined, after nine months' observation, no material change was apparent in the patient's physical condition. He was referred for a psychiatric opinion to Dr. Ivan Leveson, who reported as follows:—

> "He complains that he finds mental effort difficult, is indecisive and unable to understand things as well as previously. Prior to his illness he was very active and sociable, whereas now he has no inclination to mix with his friends, and lacks interest. He finds himself frequently thinking about his hallucinatory experiences, and the fact that he 'saw' men in his wife's bedroom, during the acute phase of his illness, has led him to doubt her fidelity. During examination he is pleasant, frank, cooperative and coherent, and there is no evidence of any acute emotional disorder."

The Wechsler-Bellevue intelligence test and the Shipley-Hartford test gave confirmatory evidence of intellectual deterioration. Electroencephalography revealed no abnormality.

TABLE 1

SUMMARY OF ANALYTICAL DATA

Days after Admission	C.S.F. (mg. Pb in 100 g)	Blood (mg. Pb in 100 g)	Urine				
			Daily Output (ml.)	S.G.	Mg. Pb/litre	Mg. Pb/litre converted to S.G. of 1024	Daily Urinary Excretion Pb (mg.)
6th	0·06	0·33	—	—	—	—	—
7th	—	—	1800	1025	0·50	0·48	0·9
8th	—	—	1600	1016	0·25	0·38	0·4
9th	—	—	1780	1014	0·28	0·48	0·5
13th	0·04	—	—	—	—	—	—
15th	0·04	—	—	—	—	—	—
22nd	0·01	0·18	2320	1013	0·16	0·30	0·38
25th	—	—	1800	1017	0·21	0·30	0·38
27th	0·01	0·12	—	—	—	—	—
34th	—	—	2270	1014	0·11	0·19	0·25
35th	—	—	1920	1014	0·13	0·22	0·25
37th	Nil	—	—	—	—	—	—
46th	—	—	2090	1012	0·11	0·22	0·23
47th	Nil	0·10	—	—	—	—	—
62nd	—	—	1330	1017	0·15	0·21	0·2
185th	Nil	—	1250	1013	0·10	0·18	0·13
186th	—	0·12	2000	1009	0·07	0·19	0·14
196th	—	0·13	—	—	—	—	—

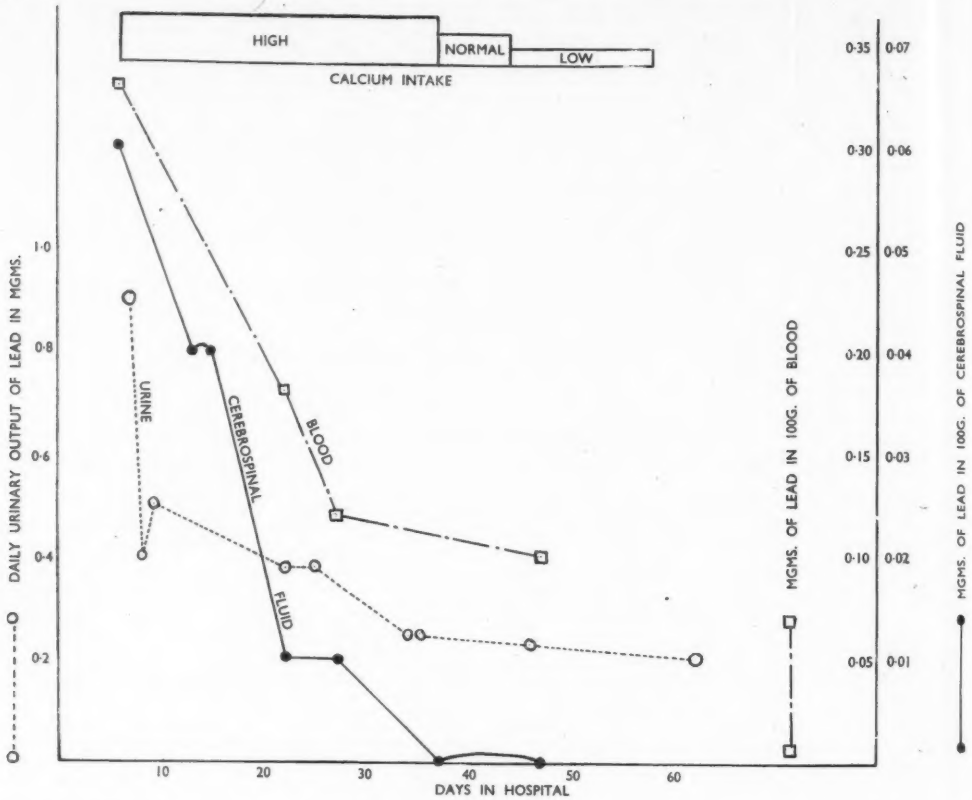

FIGURE 1.

FIG. 1.—Graph showing the daily output of lead in the urine and the concentration of lead in the blood and cerebrospinal fluid during period in hospital.

Lead Analysis

Collection of Specimens.—All specimens for analysis were collected in lead-free containers. On the seventh, eighth, and ninth nights the patient was confused and the possibility that urine collected during this period was contaminated cannot be excluded. Thereafter he was scrupulously careful. For lumbar puncture and venepuncture new autoclaved needles and syringes were used, but these were not treated to remove traces of lead. As a check, therefore, samples of blood on the twenty-second and one hundred and ninety-sixth days, and of cerebrospinal fluid on the twenty-second day, were taken with syringes and needles previously treated with nitric acid. The results of analysis of these specimens were in general conformity with those obtained by the less scrupulous technique. This finding, the absence of sudden unexplained variations and the low figures eventually obtained, lead one to suppose that no material contamination occurred.

To obviate the variations in urinary lead concentration which take place throughout any 24-hour period, analysis was carried out on the whole 24-hour specimen. The mg. per litre figure thus obtained was also converted to a standard specific gravity of 1024, as recommended by Levine and Fahy (1945). The total daily urinary excretion of lead was calculated, as this appears to afford the most satisfactory data for following the progress of a case of poisoning.

Air samples were collected by means of a hand-operated pump, and absorption in acetic acid.

Method of Analysis.—Analysis of all specimens was carried out by a dithizone method based on that described by Snyder (1947). The figures for lead concentration in cerebrospinal fluid and blood are accurate to within 0·005 mg. per 100 g., and in urine to within 0·005 mg. per litre. For this reason results recorded as "nil" in the cerebrospinal fluid should, to be exact, be stated as "less than 0·005 mg. of lead per 100 g."

Conditions in the Cooperage

Since a severe lead hazard is now uncommon in industry in this country, a more detailed description of the cooperage is important.

It was in 1940, when the shortage of timber made this profitable, that the firm first undertook the repair of barrels which had contained white lead. They failed to give notification that the work was in progress, and therefore evaded inspection.

The building, a ramshackle barnlike structure, was of the most primitive nature. Apart from large open doors, and windows high up in the walls, there was no system of ventilation. The floor, which was rough and pitted, was dry-swept. There were no washing facilities and no canteen, and although they did not eat on the premises the men smoked when they liked and drank tea periodically. Each man wore an apron over his ordinary clothes. The dust of white lead was evident everywhere, particularly on the men's hands, hair, and clothing.

Most of the barrels had contained dry white lead. Before cooping these the men had instructions to don masks, damp the barrel, empty out any white lead in the bottom, and replace the lid. Depending on the cooper himself, most or all of these precautions were disregarded; when the work was carelessly performed clouds of dust rose up from the barrels and from the floor around them. Even more dangerous were barrels which had contained white lead ground in linseed oil; this had set hard and to soften it a small wood fire was lighted in the bottom of the barrel, which after a few minutes could be scraped clean. Perhaps the most serious hazard was a small brazier, on which an apprentice was heating a kettle for the coopers' cup of tea; for fuel he was using parts of a barrel thick with ground white lead. This was probably responsible for the high lead content of the surrounding air when no work was in progress.

Analyses of atmosphere samples gave the following figures:—

(1) General air sample some 5 yds. from brazier (no active work in progress): 20 mg. lead per 10 cu.m.

(2) Air at face level, when working on white lead barrel with prescribed precautions: 50 mg. lead per 10 cu.m.

(3) Same barrels, but working with extreme carelessness: 660 mg. lead per 10 cu.m.

(4) Air at face level, when working on ground white lead barrel, after heating in the usual manner: 830 mg. lead per 10 cu.m.

(5) Fumes issuing from brazier: 1,300 mg. lead per 10 cu.m.

Lane (1936) has suggested that the maximum permitted contamination of the atmosphere with lead be fixed at 2 mg. per 10 cu.m., and this figure, or slightly less, is generally accepted in Great Britain and in the United States of America.

Three coopers and two apprentices were employed. The coopers performed identical work with apparently similar risks; the apprentices were given lighter and less hazardous tasks. The apprentices had not been long at the job; they were well, they had no lead line and their blood was normal. All three coopers showed a blue line on the gum margins. Two, who had been working for ten years on lead barrels, had no symptoms of intoxication, and although they looked pale, their hæmoglobin was over 90% (Haldane); one had a stippled count of 4,000, the other of 400 per million red blood cells. The third cooper had been working on the barrels for three years. He had suffered from periodic constipation and colic; he looked pale and his blood count showed hæmoglobin 9·7 g. (65% Haldane); reticulocytes 3·5%; stippled cells 4,000 per million red blood cells.

We were informed that our patient had been an extremely careless worker. He took none of the prescribed precautions, and in particular was in the habit of rolling his own cigarettes when his hands were covered with white lead.

Discussion

Predisposing Factors.—Of four coopers, exposed to apparently similar risks, two developed lead poisoning and two remained in good health. Personal idiosyncrasy may be postulated to explain this fact, but it is difficult to prove. Alcoholics are particularly susceptible to lead poisoning, and the effect on the brain of alcohol or a vitamin B deficiency might well render it more vulnerable to the toxic action of lead. The man with encephalopathy was an alcoholic, but his mate, an even heavier drinker than he, was perfectly well after ten years' exposure, and the other cooper who showed evidence of intoxication was a teetotaller. Kehoe (1947) stresses "the generally unrecognized variability of lead exposure from person to person and from time to time in the same occupation". Atmospheric samples taken during work in the cooperage showed that extreme carelessness increased the lead concentration tenfold; this finding alone might account for apparent differences in susceptibility among the coopers.

Diagnosis.—The possibility of lead poisoning in this case was at first overlooked, through failure to obtain a good history and to insist on a detailed examination of a confused and uncooperative patient.

But even when it is realized that a patient has been exposed to lead, the diagnosis of encephalopathy presents considerable difficulty. The condition is so uncommon that cerebral symptoms in lead workers are much more likely to be due to causes other than lead. High concentrations of lead in the blood and in the urine are proof only of absorption, not of intoxication, but normal or low levels are of value in excluding the diagnosis, since severe absorption is necessary to produce encephalopathy. In the present state of our knowledge the diagnostic significance of lead in the cerebrospinal fluid is doubtful. There appears to be no definite relationship between the concentration in the cerebrospinal fluid and symptoms of encephalopathy, a point illustrated in this case by the absence of lead during relapse.

The clinical picture of encephalopathy is not distinctive. In its various forms it is closely simulated by hypertensive encephalopathy, uræmia, idiopathic epilepsy, neurosyphilis, pellagra without cutaneous manifestations, Wernicke's encephalopathy, delirium tremens and other causes of severe delirium, cerebral tumour, encephalitis, meningitis, and intoxications with various drugs.

Although in our case there was eventually ample proof of absorption of lead, and also of intoxication as evidenced by the blood changes, the possibility that the cerebral condition was due to other causes had to be seriously entertained.

Headache, vomiting, and papillœdema, and the progressive nature of his symptoms suggested a "space-occupying" lesion, and the history of cough, hæmoptysis, and loss of weight, with beaking of the finger nails, pointed to a primary lesion in the lungs. The close resemblance to cerebral tumour has been noted in many accounts of encephalopathy.

Wernickes' encephalopathy was also considered. Rapid improvement without the exhibition of any of the vitamin B group, and the normal blood pyruvic acid just before and after relapse, rendered this diagnosis unlikely.

Delirium tremens was the only other condition which presented a real problem in diagnosis. The history of cerebral symptoms over some months, the absence of tachycardia, pyrexia, leucocytosis or severe constitutional symptoms, and the presence of definite papillœdema were the main differentiating features.

Clinical Features.—The case described here was, in the main, characteristic of the delirious type of lead encephalopathy, but certain features merit further discussion.

Arterial Hypertension.—According to Traube (1878) and Vaquez (1904) hypertension is an invariable accompaniment of lead encephalopathy, and Cantarow and Trumper (1944) concluded that many, if not indeed the majority, of the phenomena of lead encephalopathy are due to hypertension, particularly the convulsive manifestations and the subjective and objective ocular abnormalities.

A sufficient number of cases of lead encephalopathy with normal blood pressures have now been recorded to disprove the contentions of Traube and Vaquez, and the case reports of Hamilton (1925), Evans (1932), Chalmers (1940), and Nussey and Drybrough-Smith (1940) show clearly that all the major manifestations of encephalopathy, including coma, delirium, convulsions, and transient visual disturbances and aphasia, may occur in the absence of hypertension. Evans noted constriction of the retinal arteries in the periods between convulsions, when the blood pressure was 120/70 mm. Hg and attacks of severe headache were followed by temporary homonymous hemianopia. Our patient experienced transient blindness once, and a temporary weakness of both hands on two occasions, but at no time was the blood pressure found to be raised.

It seems likely that arterial spasm is the underlying cause of the transient pareses, aphasia and amaurosis which occur in hypertensive and lead encephalopathy, as is the case in the similar phenomena associated with migraine. It is interesting that they have not been observed in poisoning with tetra-ethyl lead, which appears to act directly on the nervous tissue rather than on the vessels. Fishberg (1939) has suggested that, in the instances of lead encephalopathy without hypertension, "it would seem plausible, though not proved, that the vasoconstriction is largely confined to the cerebral vessels without sufficient splanchnic and other vasoconstriction to produce hypertension".

Cerebrospinal Fluid.—From his study of cases in children, McKhann (1932) concluded that the signs of lead encephalopathy were pathognomonic, not of lead poisoning, but only of increased intracranial pressure. While it is improbable that raised intracranial pressure can account for all the manifestations of encephalopathy, particularly the transient phenomena just discussed, the constant relationship between cerebral symptoms and increased cerebrospinal fluid pressure was a noticeable feature of our case; a fall in pressure accompanied the initial clinical improvement, and a rise occurred during relapses.

Fig. 1 shows an approximate parallel between the concentration of lead in the blood and cerebrospinal fluid, although the infrequency of the estimations probably gives an artificial stability to the

curves. It is interesting that Straube (1948) has recently demonstrated a similar relationship between the content of lead in the blood and cerebrospinal fluid in lambs given increasing doses of lead acetate.

Hæmoptysis.—The cause of hæmoptysis in this case was not revealed, but may have been associated with chronic bronchitis. There appears to be no conclusive evidence that lead causes specific lesions in the respiratory organs. The finger clubbing was also unexplained.

Relapse.—This does not appear to be common during recovery from encephalopathy due to inorganic lead, although it is not infrequently seen in tetra-ethyl lead poisoning (Machle, 1935). Alice Hamilton (1925) records a case similar to ours, though more severe.

Treatment.—The symptomatic treatment of lead encephalopathy includes lumbar puncture and injections of hypertonic salines to lower intracranial pressure, sedatives (in particular the barbiturates) for the control of convulsions and mental excitement, and measures to combat dehydration and starvation. In children, Bucy and Buchanan (1935) and other workers have recommended decompression for the relief of raised intracranial pressure and to reduce the likelihood of complications. The curative treatment of lead poisoning is the subject of much controversy. Whilst no workers question the beneficial effect of calcium in lead colic, due at least in part to a local action on the intestinal muscle, several authorities express doubt as to its value in other forms of lead poisoning. In the case of encephalopathy described in this paper, a high calcium and phosphorus diet, with additional calcium intravenously and by mouth, was started on the sixth day in hospital. By this time the patient had been free from exposure for over a week, and the bowel frequently purged with magnesium sulphate. The rapid decrease in urinary lead excretion, evident from the seventh to the ninth days, may have been taking place for some days previously. Following the administration of calcium there was undoubtedly marked clinical improvement, but this improvement ran parallel with the fall in cerebrospinal fluid pressure, and may have been due, at any rate in part, to repeated lumbar puncture. A normal calcium intake during the sixth week, and subsequently a low calcium diet, were not accompanied by any rise in the lead content of the blood, cerebrospinal fluid, or urine. There was, it is true, a return of headache and a rise in the pressure of the cerebrospinal fluid during the sixth week, but these did not recur when a low calcium diet was given, and a much more serious relapse had taken place when the patient was receiving large amounts of calcium. No definite conclusions can therefore be drawn regarding the effect, if any, of the administration of calcium in this case. While under treatment in hospital my patient's progress, as judged by the concentration of lead in the blood and urine, was essentially similar to that described by Kehoe, Thamann, and Cholak (1933) in patients to whom no calcium was given. Later, although the concentration of lead in the urine continued to fall, that in the blood remained disturbingly high; it was actually greater on the one hundred and ninety-sixth day than on the twenty-seventh (Table I). This was considered a contraindication to any measures designed to mobilize "stored" lead.

The one therapeutic measure which appeared to be of undoubted value was the repeated withdrawal of cerebrospinal fluid by lumbar puncture.

The number of recorded cases of inorganic lead encephalopathy in adults, treated by various modern methods, is too small for statistical analysis of results to be of much value.

Summary

A cooperage is described in which a severe lead hazard caused two cases of lead poisoning; one patient developed encephalopathy.

The syndrome of lead encephalopathy in adults is reviewed. The difficulties in diagnosis, the possible causes of apparent susceptibility to lead, the absence of hypertension in this and other cases, and the treatment of encephalopathy are discussed. Some of the essential differences between this form of encephalopathy and that due to tetra-ethyl lead are outlined.

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THE SIZE AND NATURE OF DUST PARTICLES FOUND IN LUNG TISSUE

BY

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Ten years ago, when we were making a physical study of the dust hazard in south Wales anthracite mines as part of the Medical Research Council's early investigation of chronic pulmonary disease in coal miners, we had the opportunity of examining the dust particles on two slides containing sections of a lung of a man who had died from silicosis after many years' work as a shot-firer in one of the anthracite mines included in our study.

Of the two lung sections, one had been stained but was otherwise untreated, and the other had been incinerated and the soluble ash removed by treatment with hydrochloric acid. Passing reference to these observations has already been made (Bedford and Warner, 1943), but in view of the present renewed interest in particle size in relation to pneumoconiosis it seems desirable to record our data in greater detail.

Method of Examination

The sections were examined by a technique similar to that which we employed in the evaluation of samples of air-borne dust obtained with a thermal precipitator. The microscope had a 2 mm. oil-immersion apochromatic objective, N.A. 1·37, and a X15 eyepiece, giving an overall magnification of about 1,200. The source of illumination was a 100 c.p. "pointolite" lamp with a blue-green filter. With these optical arrangements dust particles of rather less than 0·2 micron diameter could be discerned in the nodules and reticulation areas as discrete entities. For the measurement of the size of the dust particles an eyepiece graticule of the type described by Patterson and Cawood (1936) was used. When particles were massed together in an aggregate efforts were made to count and measure the individual particles comprising the aggregate.

On the untreated section counts were made on three typical nodules and three typical reticulation areas. Each of these areas was indicated to us by our colleague Dr. D'Arcy Hart. Strips across each of these six areas were examined and in each area not less than 2,000 dust particles were measured. All particles greater than 0·4 micron in diameter were classified into the groups "coal" and "minerals other than coal".

Coal particles and any acid-soluble ash had been removed from the other slide by the processes of incineration and acid treatment, so it was expected that the size distributions of dust particles on this slide would agree more or less closely with those of the particles of minerals other than coal on the untreated section. On this treated slide Dr. D'Arcy Hart identified two areas —one a nodule and one a reticulation area—which had been examined on the untreated section, and particle-size distributions were ascertained for these areas.

We have referred to the classification of dust particles accordingly as they were of coal or of other minerals. Early in our study of air-borne dusts it had appeared to us that in the thermal precipitator records it was possible to discriminate between the coal and other particles, for what we interpreted to be anthracite particles, even down to very small sizes, were quite black, whereas those of other minerals were not black and had a glossy appearance. Tests were made of the validity of this discrimination, and the results justified the visual discrimination of coal from other minerals in the examination of our samples (Bedford and Warner, 1943). The distinction was therefore made as a matter of routine. Many particles less than 0·4 micron in size could be so classified, but in our routine examinations only particles of 0·4 micron or greater diameter were sub-divided into the two groups.

In our examination of the untreated lung section particles of coal and of other minerals were differentiated in the same way. All the dust seen in the incinerated section was "non-coal", and since about half the visible particles in that section were smaller than 0·4 micron it seemed desirable to attempt to estimate the numbers of small particles of minerals other than coal in the untreated section.

As we had observed with air-borne dust, in the dust deposits in the lung sections the ratio of the number of coal to the number of non-coal particles decreased rapidly with decreasing particle size. At sizes below about 2 microns the logarithm of the ratio of coal to non-coal particles diminished in proportion to the particle size. Thus, in the case of the one nodule which was examined on both the treated and the untreated sections, in the untreated section the coal to non-coal

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ratios at sizes of 0·4 to 0·8 microns, 0·8 to 1·2 microns, 1·2 to 1·6 microns, and 1·6 to 2·0 microns, were respectively 0·85, 2·23, 9·47, and 56·0. The logarithms of these ratios, plotted against the mean particle size fell very closely about a straight line, and it seemed likely that extrapolation for a short distance to smaller sizes was probably not a very hazardous procedure. The probable ratios of coal to non-coal particles were therefore estimated in that way, and the total counts of particles of under 0·2 microns and of 0·2 to 0·4 microns diameter were subdivided into coal and non-coal fractions on that basis.

The Sizes of Dust Particles found in Lung Tissue

Table 1 gives a comparison of the size distributions of dust particles measured in the selected (*a*) nodule and (*b*) reticulation area in the incinerated section with the size distributions of non-coal particles in the same areas of the untreated section.

Scrutiny of Table 1 shows that the percentage size distributions of the particles in the untreated section larger than 0·4 micron, and visually discriminated as minerals other than coal, are very similar to the

TABLE 1

COMPARISON OF SIZE DISTRIBUTIONS OF DUST PARTICLES IN INCINERATED AND ACID-TREATED SECTION AND OF NON-COAL PARTICLES IN UNTREATED SECTION

	Total	Particles of Size (microns)										
		Under 0·2	0·2 to 0·4	0·4 to 0·8	0·8 to 1·2	1·2 to 1·6	1·6 to 2·0	2·0 to 2·5	2·5 to 3·0	3·0 to 4·0	4·0 to 5·0	5·0 and over
I. Nodule												
Incinerated and acid-treated section												
Particles counted ..	1000	288	256	357	87	10	2	—	—	—	—	—
Untreated section												
Particles of minerals other than coal actually counted ..	412	—	—	300	93	17	2	—	—	—	—	—
Estimated, below 0·4 micron	454	210	244									
Probable totals, minerals other than coal	866	210	244	300	93	17	2	—	—	—	—	—
Percentage size distributions												
Incinerated and acid-treated section ..	100·0	28·8	25·6	35·7	8·7	1·0	0·2	—	—	—	—	—
Minerals other than coal in untreated section	100·0	24·2	28·2	34·7	10·7	2·0	0·2	—	—	—	—	—
II. Reticulation area												
Incinerated and acid-treated section												
Particles counted ..	1011	219	306	345	63	36	25	8	5	4	—	—
Untreated section												
Particles of minerals other than coal actually counted ..	233	—	—	168	49	12	4	—	—	—	—	—
Estimated, below 0·4 micron	243	95	148									
Probable totals, minerals other than coal	476	95	148	168	49	12	4	—	—	—	—	—
Percentage size distributions												
Incinerated and acid-treated section ..	100·0	21·6	30·3	34·1	6·2	3·6	2·5	0·8	0·5	0·4	—	—
Minerals other than coal in untreated section	100·0	20·0	31·3	35·3	10·3	2·5	0·8	—	—	—	—	—

distributions of particles greater than 0·4 micron in the incinerated and acid-treated section. When, for the untreated section, the numbers of non-coal particles smaller than 0·4 micron are estimated by extrapolation, the proportions of these particles, in both the nodular and the reticulation areas, are very similar to those observed in the incinerated section. Hence the comparable percentage size distributions in Table 1 based on the numbers of particles of all sizes, counted or estimated, closely resemble each other.

In the untreated section, the size distributions of the dust particles measured in different nodules were found to be very similar, and the same was true of the size distributions for the different reticulation areas examined. Table 2 shows the size distributions of all the dust particles measured in the untreated section, without regard to the nature of the dust.

TABLE 3

TOTALS OF PARTICLES LARGER THAN 0·4 MICRON COUNTED IN 3 NODULES AND 3 RETICULATION AREAS OF UNTREATED SECTION OF LUNG

	No. of Particles			Percentage		
	Coal	Non-coal	Total	Coal	Non-coal	Total
Nodules ..	3627	968	4595	78·9	21·1	100·0
Reticulation areas ..	4299	715	5014	85·7	14·3	100·0

coal was 21·1% in the nodules, and only 14·3% in the reticulation areas.

Table 4 gives the estimated numbers of particles of the two kinds of all sizes, the sub-division of the particles smaller than 0·4 micron having been carried

TABLE 2

SIZE DISTRIBUTIONS OF DUST PARTICLES (TOTAL DUST) IN UNTREATED SECTION

	Total	Particles of Size (microns)										
		Under 0·2	0·2 to 0·4	0·4 to 0·8	0·8 to 1·2	1·2 to 1·6	1·6 to 2·0	2·0 to 2·5	2·5 to 3·0	3·0 to 4·0	4·0 to 5·0	5·0 and over
Particles counted												
Nodules	6059	565	899	1553	945	568	516	374	261	275	86	17
Reticulation areas ..	6112	370	728	1476	1124	642	578	446	258	312	153	25
Percentage size distributions												
Nodules	100·0	9·3	14·8	25·7	15·6	9·4	8·5	6·2	4·3	4·5	1·4	0·3
Reticulation areas ..	100·0	6·1	11·9	24·1	18·4	10·6	9·4	7·3	4·2	5·1	2·5	0·4

The distributions for nodules and for reticulation areas are very similar, but the nodules contained the slightly larger proportion of very small particles. It will be shown later that differences in the proportions of coal and other particles account for this difference in the size distributions of the whole dust.

Of all the particles measured, 0·3 or 0·4% were larger than 5 microns. If any of these were aggregations of smaller particles we failed to discern it. Seventeen such particles were measured in nodules, and of these 15 were 6 microns in diameter, and the remaining two were 8 microns in size. Of the 25 particles larger than 5 microns measured in reticulation areas, 22 were 6 microns and three were 8 microns in diameter.

The Nature of the Dust

The numbers of particles of coal and of minerals other than coal discriminated visually down to a diameter of 0·4 micron are shown in Table 3.

The proportion of particles of minerals other than

out by the extrapolation method previously described.

Here, again, the proportion of non-coal particles in the nodules is about one and a half times as large as in the reticulation areas, but the actual percentages of non-coal particles are distinctly higher.

The size distributions of particles of coal and of other minerals are given in Table 5. The subdivision

TABLE 4

TOTALS OF PARTICLES OF ALL SIZES COUNTED AND ESTIMATED IN NODULES AND RETICULATION AREAS OF UNTREATED SECTION

	No. of Particles			Percentage		
	Coal	Non-coal	Total	Coal	Non-coal	Total
Nodules ..	4004	2055	6059	66·1	33·9	100·0
Reticulation areas ..	4726	1386	6112	77·3	22·7	100·0

TABLE 5

SIZE DISTRIBUTIONS OF PARTICLES OF COAL AND OF OTHER MINERALS* FOUND IN NODULES AND RETICULATION AREAS IN UNTREATED SECTION

	Total	Particles of Size (microns)										
		Under 0·2	0·2 to 0·4	0·4 to 0·8	0·8 to 1·2	1·2 to 1·6	1·6 to 2·0	2·0 to 2·5	2·5 to 3·0	3·0 to 4·0	4·0 to 5·0	5·0 and over
I. Numbers of Particles Counted												
1. *In Nodules*												
Coal	4004	117	260	840	731	531	512	374	261	275	86	17
Minerals other than coal	2055	448	639	713	214	37	4	—	—	—	—	—
2. *In Reticulation areas*												
Coal	4726	122	305	951	976	609	570	445	258	312	153	25
Minerals other than coal..	1386	248	423	525	148	33	8	1	—	—	—	—
II. Percentage Distributions												
1. *In Nodules*												
Coal	100·0	2·9	6·5	21·0	18·3	13·3	12·8	9·3	6·5	6·9	2·1	0·4
Minerals other than coal	100·0	21·8	31·1	34·7	10·4	1·8	0·2	—	—	—	—	—
2. *In Reticulation areas*												
Coal	100·0	2·6	6·5	20·1	20·6	12·9	12·1	9·4	5·5	6·6	3·2	0·5
Minerals other than coal	100·0	17·9	30·5	37·9	10·7	2·3	0·6	0·1	—	—	—	—

*Subdivision of particles less than 0·4 micron into coal and other minerals estimated.

of particles below 0·4 micron in size into coal and other minerals has been estimated.

The percentage size distributions of the coal particles found in nodules and in reticulation areas are practically identical, and the same applies to particles of minerals other than coal. As in the dust clouds in mine air, the larger particles are of coal, while it is estimated that of the non-coal particles 86 or 87% were less than 0·8 micron in diameter.

The difference between the size distributions of the whole dust found in nodules and in reticulation areas, which are noted in Table 2, are due to the different proportions of the two fractions of the dust present. The dust in the reticulation areas contains relatively more coal and less minerals other than coal than is found in nodules. It is not for us to discuss how this has arisen, but since the size distributions of the two fractions of dust are the same at the two sites there appears to have been no discrimination as to the size of particles conveyed.

Pathological Significance of Particle Size

As a solid material is disintegrated into smaller and yet smaller particles, the surface area of a given weight of the solid is much increased, so that those properties which depend upon the activity of the surface molecules are greatly enhanced. Very large dust particles are probably, therefore, of little pathological significance. There is, however, some difference of opinion as to the maximum particle size which can be considered to be of pathological importance. Gardner (1939) reported :

> " We have injected a series of doses of silica particles of different sizes having comparable surface areas. Regardless of the total surface area the silica-significant reaction only occurs when the particles are 3 microns or less in diameter ".

It seems to be fairly generally accepted by American workers that in estimating the dust hazard particles larger than about 3 microns can be ignored, and Sayers and Jones (1938) mention that the majority of particles found in the lung are 1 to 3 microns in size.

In our examination of lung sections we found dust particles as large as 8 microns, but they were of coal. In the non-coal fraction of the dust in our specimens (which fraction presumably contains all or virtually all of the siliceous material) no particles larger than 4 microns were noted, but the number of particles of minerals other than coal larger than 4 microns present in the air of an anthracite mine is small. Gardner (1939) remarks that particles as large as 10 microns are found in human lungs.

We made many observations of air-borne dust at the colliery where the man from whom our specimens were taken had worked for many years.

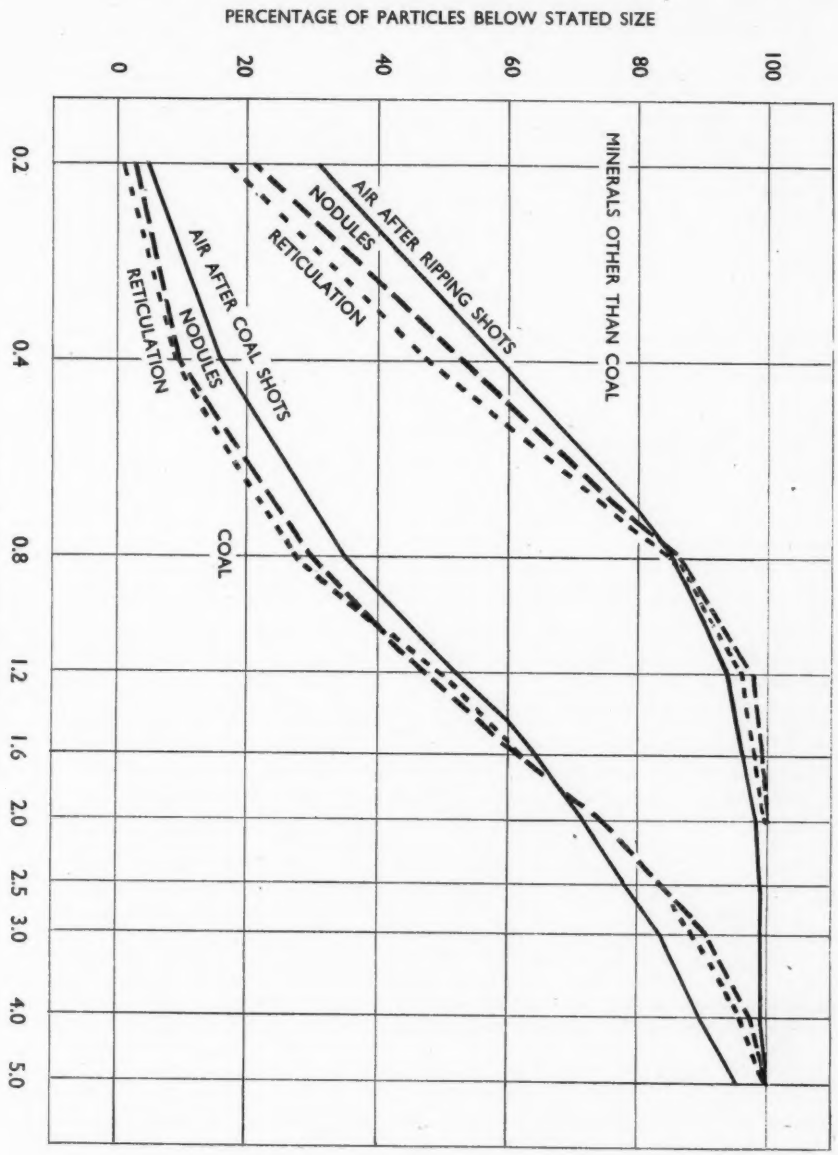

FIG. 1.—Particle size distributions of dust in mine air and in lung tissue.

The size distributions of non-coal particles found in the lung sections were rather similar to the distributions of non-coal particles in the air-borne dust after ripping shots had been fired in stall roads, and the coal particles found in the lung tissue had size distributions not unlike the distribution of coal-dust particles after shots had been fired in coal.

In Table 6 the particle size distribution of the dust of minerals other than coal found in the air after ripping shots had been fired is compared with the distributions of such dust found in lung sections; and, similarly, the size distribution of air-borne coal dust at the coal-face after shots had been fired in the coal is compared with the distributions of coal dust in the lung tissue. The classification of the particles in the lowest two size-groups as coal or as other minerals has been done by the method described earlier. The data are also shown graphically in Fig. 1.

TABLE 6

COMPARISON OF PARTICLE SIZE DISTRIBUTIONS OF DUST IN MINE AIR AND IN LUNG TISSUE

Dust	Percentage of Particles of Diameter Less than (microns)									
	0·2	0·4	0·8	1·2	1·6	2·0	2·5	3·0	4·0	5·0
Minerals other than coal										
In colliery air after ripping shots ..	31·2	59·5	85·9	94·9	96·5	98·4	99·0	99·0	99·0	100·0
In lung tissue (nodules)	21·8	52·9	87·6	98·0	99·8	100·0	—	—	—	—
,, ,, (reticulation areas) ..	17·9	48·4	86·3	97·0	99·3	99·9	100·0			
Coal										
In colliery air after shots in coal ..	4·7	16·6	35·9	52·9	64·7	72·2	78·1	83·9	90·1	94·8
In lung tissue (nodules)	2·9	9·4	30·4	48·7	62·0	74·8	84·1	90·6	97·5	99·6
,, ,, (reticulation areas) ..	2·6	9·1	29·2	49·8	62·7	74·8	84·2	89·7	96·3	99·5

The size distributions of the dust in the lung sections are similar in type to those of the air-borne dust after shot-firing, i.e., at the times when the highest dust concentrations were likely to have been encountered. In the lung sections the fraction of the dust containing siliceous material contained no large particles, but large particles of minerals other than coal were exceedingly rare in the air-borne dust. In the lung sections the proportions of finer particles in both the coal and the non-coal fractions of the dust were lower than in the air-borne dust, and this is in general accord with the known facts concerning the retention of fine dust in the lungs.

In a recent paper, Brown, Cook, Ney, and Hatch (1950) report the results of a study of the influence of particle size upon the retention of particulate matter (china clay) in the human lung, and they give the calculated upper and lower limits of alveolar deposition of particles of various sizes. Alveolar deposition is at a maximum (about 55%) for particles of 1 micron diameter, while between 8 and 23% of the 5-micron particles inhaled are retained in the alveoli.

From the curves given by Brown and his colleagues we have computed the limits of the percentage size distributions of the inhaled dusts that might have been expected to give rise to deposits with size distributions such as we found in reticulation areas. In these calculations it has been assumed that our non-coal particles had a density equal to that of china clay, and that the density of our coal particles was one-half that of china clay. The computed size distributions are shown in broken lines in Fig. 2, for both the coal and the non-coal fractions of the dust. Fig. 2 also shows the size distributions of non-coal particles observed in the mine air after ripping shots had been fired; and the size distributions of coal particles observed (*a*) after shots had been fired in coal, and (*b*) in the air of gate roads under ordinary travelling conditions when no shots had recently been fired. The distribution of non-coal particles observed in the mine air after ripping shots had been fired, i.e., when the shot-firer probably is most severely exposed to non-coal dust, is remarkably similar to the computed distribution. The computed size distributions for the coal fraction of the dust are very similar to the observed distributions of air-borne coal particles under normal conditions in gate roads, where the shot-firer spends much of his time walking from one working place to another, and at the coal face after shots had been fired in coal. If these two observed distributions were averaged the resultant mean distribution would be entirely within the calculated limits. Belt and Ferris (1942) remarked that the collier's lung is in a very real sense his occupational log book, and our data appear to give support to that statement.

There is one other point that seems worthy of mention. During a discussion of our data with colleagues it was suggested that the similarity between the sizes of particles in nodules and in reticulation areas seemed to indicate that no notable

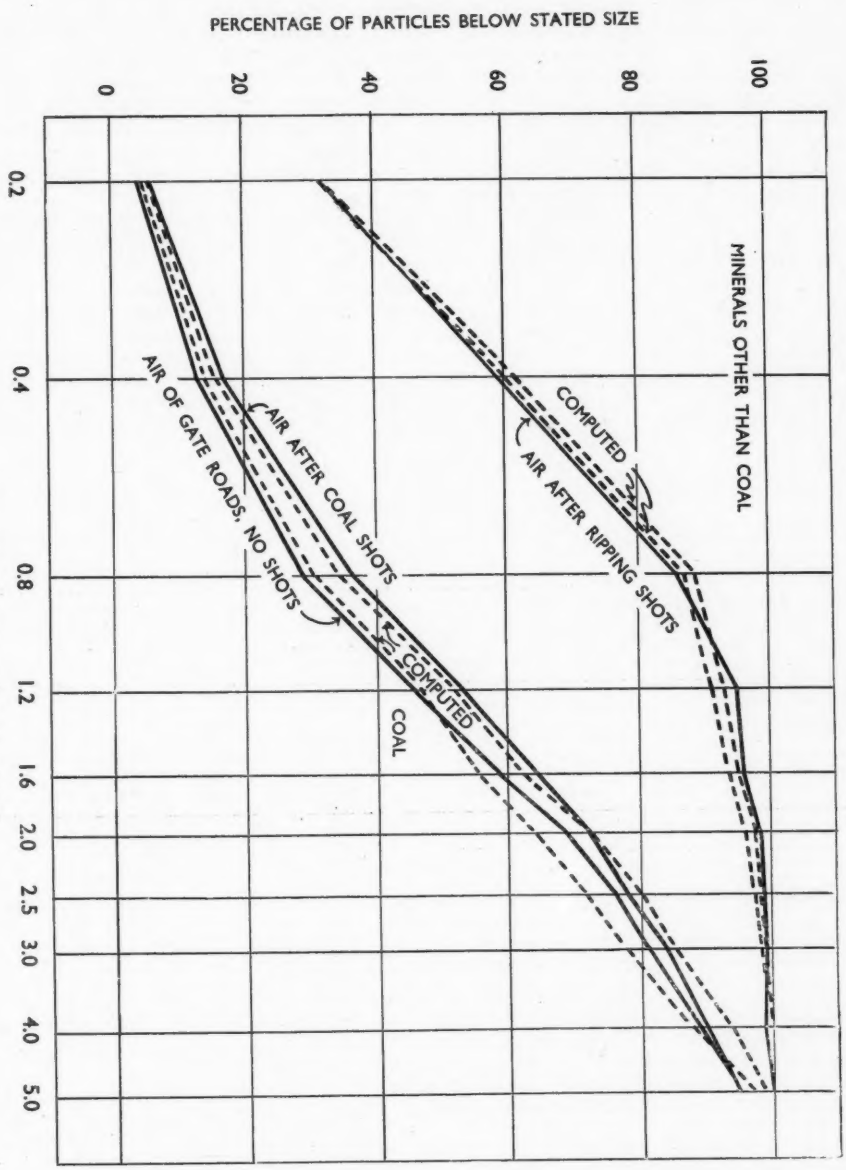

Fig. 2.—Computed particle size distributions (from data of Brown, Cook, Ney, and Hatch, 1950) of inhaled dust which would be expected to lead to alveolar retention of dust with the size distributions observed in reticulation areas compared with observed size distributions of air-borne dust particles.

reduction in size occurred after particles had resided in nodules for a period of years, and that the question might therefore arise whether the nodule in the coal miner is a foreign body reaction rather than a tissue reaction aroused by the solution of mineral matter. We doubted whether such an interpretation could be attached to our data, and as a rough test we estimated from the percentage size distribution of the non-coal particles found in reticulation areas (Table 5) what would be the probable size distribution of the remaining dust if some solution occurred. Although solubility is a function of particle size we assumed that from all the particles the outer layer would be dissolved for a uniform thickness of 0·025 micron, 0·05 micron, and 0·10 micron ; that is, we assumed reductions of the diameters of the particles of 0·05, 0·1, and 0·2 micron.

The results of these calculations are shown in Table 7. With each degree of solution assumed some of the smallest particles would be dissolved completely or would become too small to be seen by our microscope technique, and allowance has been made for that effect. With solution of a layer 0·1 micron in thickness, we estimate that the weight of the dust would be reduced by about one-half. Yet in spite of this the computed size distributions are virtually the same as that of the original dust in the reticulation areas. Thus, our data should not be taken to indicate that no solution occurs after the dust particles are deposited in the lung tissue.

Size distributions are given separately for particles of coal and of minerals other than coal.

Particles were examined (*a*) in nodules, and (*b*) in reticulation areas. Of all the particles measured 0·3 to 0·4% were larger than 5 microns, and particles as large as 8 microns were found in both nodules and reticulation areas. As in the dust clouds in mine air, the larger particles were of coal. Of the non-coal particles 86 or 87% were less than 0·8 micron in diameter.

The size distributions of the dust in the lung sections were similar in type to those of the air-borne dust, after shot-firing, at the colliery where the man had worked, although the proportion of very small particles was somewhat smaller in the lung sections than in the air-borne dust. It is shown that when allowance is made for the effects of particle size on alveolar retention, the observed size distributions of the air-borne dust are very close to what would be expected to account for the size distributions of the dust particles found in the lung tissue.

TABLE 7

PERCENTAGE SIZE DISTRIBUTIONS OF PARTICLES OF MINERALS OTHER THAN COAL SHOWING ESTIMATED EFFECTS OF REDUCTION OF SIZE BY SOLUTION OF OUTER LAYERS OF PARTICLES

Range of Particle Size	Percentage of Particles of Minerals other than Coal within Given Range of Particle Size				
	Observed in Reticulation Areas	Estimated after Assumed Loss by Solution of a Layer of Thickness of			Observed in Nodules
		0·025 micron	0·05 micron	0·10 micron	
Below 0·4 microns	48·4	48·1	48·1	51·3	52·9
,, 0·8 ,, ...	86·3	85·7	86·5	88·2	87·6
,, 1·2 ,, ...	97·0	96·8	96·9	97·4	98·0
,, 1·6 ,, ...	99·3	99·3	99·4	99·6	99·8
,, 2·0 ,, ...	99·9	99·9	100·0	100·0	100·0
,, 2·5 ,, ...	100·0	100·0	100·0	100·0	100·0
Percentage reduction of volume (or weight) of dust	—	16	33	53	—

In nodules and in reticulation areas the particle size distributions were very similar. Rough calculations of the effects of partial solution of the dust on particle size distribution suggest that the similarity of the size distributions should not be taken to indicate that no solution occurs after particles are deposited in lung tissue.

Summary

The paper gives detailed size distributions of dust particles found in specimens of lung tissue from a shot-firer who had died from silicosis after many years' work in an anthracite mine.

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BOOK REVIEWS

Vanadium Pentoxide Dust : A Clinical and Experimental Investigation on its Effect after Inhalation. By Sven Gösta-Sjöberg. Stockholm. 1950. Pp. 188. Price not stated.

Nearly 40 years ago Dutton, an American doctor, described a group of symptoms which he attributed to chronic poisoning with vanadium pentoxide. This group included a characteristic paroxysmal, dry, irritating cough which could lead to hæmorrhage in the lung, irritation of the nose, eyes, and throat, anorexia, nausea, diarrhœa or constipation, anæmia, cachexia, albuminuria with casts, hæmaturia (renal), tremor, headache, neuroretinitis, amaurosis, vertigo, and mental disorders. The prognosis of this apparently devastating syndrome was held to be good. About 30 years later Symanski, an observer in Germany, studied 19 cases and reduced the picture to irritation of the upper respiratory tract and of the conjunctiva, and an *x*-ray picture of chronic bronchitis sometimes with bronchiectasis. All the other phenomena alleged by Dutton were discarded. Seven years later Wyers in Great Britain gave an account of 10 cases observed in a factory in Essex. The syndrome was attributed to the inhalation of vanadium pentoxide dust and included the following : pallor of the skin (without signs of anæmia) ; greenish-black discoloration of the tongue which disappears in a few days if removed from exposure ; paroxysmal cough (rarely associated with hæmoptysis) ; dyspnœa ; pains in the chest ; palpitation on exertion ; tremor of the fingers and arms ; emphysema ; bronchitis ; bronchospasm ; raised blood pressure ; reticulation of the lung (*x*-ray) ; and a probable greater liability to colds and pneumonia. Clearly almost the whole of this syndrome relates to the respiratory tract. The alleged pallor and tremors also described by Dutton are unexplained. The discoloration of the tongue is said to occur most frequently among men with septic teeth and furred tongues. Actually Wyers mentions this sign in one only of his 10 summarized cases.

Certain features of Wyers' description must be grasped. He lays special emphasis on emphysema and reticulation of the lung ; he refers to three cases of lower lobe pneumonia of which one was fatal, and one case of middle lobe pneumonia with the later comment that " it seems likely that colds and pneumonia are of more frequent occurrence than in the general population " ; he holds " raised blood pressure and accentuated pulmonary second sound " to be an observed feature of the syndrome but examination of the figures given in his 10 illustrative cases does not appear to support this observation.

The recent findings of Sjöberg as presented in this painstaking publication are somewhat laboriously repetitive, but the result of reading this work is to leave no doubt about what he has found. He has studied 36 workers for four years in a vanadium factory which started operations in 1946 and soon experienced cases of illness. Some of these 36 workers had worked in the " old vanadium works " where conditions had been bad. Concurrently with thorough and modern clinical, chemical, radiographic, and other examinations, the author also performed experiments on rabbits using finely ground pure vanadium pentoxide, the proportion of very small particles being much greater than that found in factory dust.

Clinically Sjöberg observed slight conjunctival irritation ; mucosal irritation with nasal catarrh ; dry and irritant throat but no serious changes in larynx or trachea ; violent cough ; wheezing chest ; dyspnœa ; seven cases of pneumonia or bronchopneumonia. This author found no evidence of chronic pulmonary changes indicative of pneumoconiosis, fibrosis, or emphysema. Blood pressure was not raised. Sjöberg showed by spectrographic analysis of the ash of blood and urine that vanadium is absorbed into the circulation in quantities which cannot be regarded as negligible.

By patch tests he demonstrated that skin sensitivity to venadate occurs in some cases, but he is not dogmatic as to possible allergic factors in the respiratory symptoms.

The " disease " is acute, comes on within a week of the first exposure to dust and lasts from a few days to a few months according to the degree of exposure. The symptoms subside except for some residual chronic changes in the pharynx and some skin sensitivity.

The differential diagnosis may be difficult, for the condition simulates a simple acute respiratory infection. The temperature curves in the pneumonia cases were similar to those found in non-industrial cases.

Sjöberg points out that, in spite of the undoubted systemic absorption of vanadium in his cases, there was no definite evidence of toxic effects. It is interesting that during the period of investigation the workers gained in weight.

Animal experiments with exposure to high concentrations of vanadium pentoxide for short periods and to low concentrations for periods up to eight months showed that inhaled vanadium pentoxide may be lethal ; that it is a strong irritant to the respiratory tract producing tracheitis and bronchopneumonia which may

leave some chronic effects ; that vanadium pentoxide is absorbed systemically ; that there is a sparse deposition of vanadium pentoxide dust in the lungs since the smallest particles ($< 5\ \mu$) are (says Sjöberg) absorbed from the lungs and can no longer be seen in them after a few weeks irrespective of the period of exposure or concentration of the dust ; that there is no development of pneumoconiosis ; that vanadium pentoxide does not produce fibrosis in the lungs. The whole, or nearly the whole, picture in the exposed animals is one of an acute disease when a high concentration of dust is used. When a low concentration is used for a long period there result chronic inflammatory changes in the nasal and tracheal mucosa, some slight emphysema, and occasional atelectasis in the lungs. Some pathological changes were found in the liver but they were difficult to assess, and on the whole the author is not inclined to lay much stress on them. Even with low concentrations and long exposure the effects must be regarded as of an acute kind, the vanadium pentoxide being apparently rapidly carried away from the lungs and excreted in the urine. Clearly the animal experiments confirmed the clinical findings.

Thus, Sjöberg was unable to confirm Wyers' description of the pulmonary part of the syndrome.

The common features which remain are conjunctivitis and pneumonia. It is clear from Wyers' account, but not from the 10 cases he summarizes, that he believed he was dealing with an industrial disease in which there was the unusual combination of systemic intoxication and " dust reticulation ".

The physical properties of vanadium pentoxide are not such that long continued action *in situ* would be expected. For example, as Sjöberg showed, the solubility of this oxide in body fluids is quite high when compared with its solubility in water. In the absence of data on the particle size distribution to which Wyers' cases were exposed, it is difficult to say what would be expected to occur in the lungs. His brief description of the method of manufacture of vanadium pentoxide applied in his factory is not sufficient to indicate precisely to what the workers were exposed nor is the description of men as " vanadium workers " more enlightening. Sjöberg explains the very sparse presence of vanadium pentoxide in the lungs ($5\ \mu$ particles in pneumonic foci) of his animals exposed to the dust referred to above as due to the solution and absorption of the smallest particles or to their being easily taken up by phagocytes. There is something unsatisfactory about this for at least two reasons : the smaller the particles the less easily are they wetted, and quantitative data on the ease with which small particles of an acidic substance are taken up by phagocytes are needed before any view on this can be justified. It would be rash to say more than that the evidence thus far tends to discount the suggestion that vanadium pentoxide dust can set up a chronic fibrosing process in the lungs. But it will be necessary to follow the cases already described for a number of years before final judgment can be made. The work under review is certainly the best on the industrial toxicology of vanadium pentoxide.

M. W. GOLDBLATT

Proceedings of the Lead Hygiene Conference of the Lead Industries Association, 1948. New York. Lead Industries Association. Price 1 Dollar.

Conferences convened by industry have considerable educational value. They are attended by those with practical knowledge of industrial medicine, together with a leavening of official and academic representatives. They are not the place to present new work, but rather provide an opportunity for the discussion of some of the older problems such as, in this instance, the relation between toxicity of lead compounds and their solubility ; stipple counts or basophilic aggregation as indicators of lead absorption ; the place of lead in urine determinations in the prevention of lead poisoning.

A study of this report provides an interesting contrast between the practice of industrial hygiene in the United States and in this country. We are apt to overlook the great advantages we enjoy as a result of the work of our early pioneers. We are inclined to accept the regulations applying to the lead trades without appreciating their value. This is emphasized when we see that in many States of the U.S.A. there is no compulsory routine examination of lead workers, and that the occupier is not obliged to provide such simple provisions as washing and feeding accommodation. It is true that these precautions are carried out voluntarily by most of the large and more progressive firms, but many of the smaller establishments fall far behind in these simple preventive measures. On the other hand we can learn much from the wide application in the United States of the more accurate laboratory methods in use in the control of lead exposure.

It is refreshing to find Dr. Lanahan emphasizing the value of lead in urine estimations, not as a diagnostic measure, but as a means to prevent excessive lead absorption.

The discussion on treatment is of great interest ; the consensus of opinion agrees with that of the more experienced workers in this country, that de-leading is bad therapy and that the patient is better left to get rid of his excess lead by himself. Papers were given on the use of BAL in lead poisoning (the conclusion that it has no use in industrial medicine is what one would anticipate), on the value of porphyrin estimations in the control of industrial plumbism, and on lead-in-air standards. The presence of experts on air pollution at this conference is an interesting feature, in that it draws attention to an aspect of industrial hygiene often overlooked by those concerned only with conditions inside the factory.

The proceedings include a report of a speech by an American industrialist. Here the interest for us lies in the fact that the United States is facing some of the same problems we have had to face here ; that industrial medicine is something more than a luxury service, and that it has an important part to play in establishing good industrial relations. " The industrialist . . . should examine the philosophy of his daily life and the things he does, to see if he is contributing something to reduce the fears and bitterness which beset us."

The breadth of approach and the success of this conference reflect the organizing ability of Manfred

Bowditch, who will be remembered for his past association with the Saranac laboratories.

R. E. LANE

Modern Practice in Dermatology. Edited by G. B. Mitchell-Heggs, O.B.E., M.D., F.R.C.P. 1950. London: Butterworth & Co. Ltd. Pp. 794. Price 63*s*.

This book is magnificently produced on art paper, with many illustrations, a few in colour. It is a composite work with 41 authors all writing on different subjects of which they are authorities. It therefore suffers from all the disadvantages which such a work invariably must have. The style of the different articles varies considerably and there is a certain amount of overlapping, but the editor-in-chief is to be congratulated on the excellent way in which he has moulded his material into a whole, which is certainly a valuable reference book on skin diseases. In some chapters there are numerous references, in others none.

The section on industrial dermatitis, which is of most particular interest to the readers of this journal, is by Dr. Sibyl Horner and is written from the point of view of prevention rather than as a description of the many different kinds of skin diseases which are found in industry. These are not omitted from the book but are described in the various sections. It would be an advantage from the aspect of industrial medicine if a chapter had been devoted to a description of these conditions.

In dealing with protection, Dr. Horner pays particular attention to the selection of personnel and considers that the patch test is of considerable value. When a worker is already enrolled in an occupation protection is important, and she discusses the question of protective clothing and barrier substances. Cleanliness is, of course, of the greatest importance, and the provision of washing accommodation and soap and water help perhaps more than anything else in the prevention of skin diseases. Inspection of personnel so that the conditions may be detected at an early stage is also stressed.

This is certainly as good a reference book of dermatology as exists at the present time. K. M. A. PERRY

Report for the Year 1949 of Central Mining—Rand Mines Group Health Department. By A. J. Orenstein.

The report on the Rand Mines Group for the year 1949 gives a large number of statistics about the health of the native labour employed in these mines, together with data on hospital and feeding costs.

What the outside worker would be particularly interested in with regard to the Rand Mines is, of course, silicosis, and the report comments that the incidence and mortality rates for this disease were respectively 0·90 per 1,000 (79 cases) and 0·10 per 1,000 (9 deaths).

For the rest the report gives the incidence rate for accidents and such diseases as pneumonia, meningococcal meningitis, influenza, enteric fever, scurvy, and heat stroke. There were 220 cases of pulmonary tuberculosis (2·52 per 1,000) and 62 deaths which is 0·71 per 1,000. These figures are not remarkable in any way nor are those for the other diseases mentioned.

K. M. A. PERRY

Principles of Medical Statistics. By A. Bradford Hill. 1950. London: *The Lancet*. 5th Edit. Pp. 282. Price 10*s*. 6*d*.

To carry out any satisfactory research in medicine at the present time a knowledge of elementary statistics is almost essential. This book of Professor Bradford Hill's is so well known, and the last edition was as recent as 1948, that there is little to say about it that has not already been said. This edition is not greatly different from the previous one, though the author has omitted some paragraphs. It is an excellent book and every doctor should possess a copy.

K. M. A. PERRY

PROCEEDINGS OF THE ASSOCIATION OF INDUSTRIAL MEDICAL OFFICERS

The 59th meeting of the Association was held in the Lecture Theatre of the London School of Hygiene and Tropical Medicine. On April 28, 1950, the second biennial John C. Bridge memorial lecture entitled " Doctor and Workman " (see page 105) was given by Dr. Andrew Meiklejohn, Reader in Industrial Health at the University of Glasgow.

On April 29 a symposium was given on " Occupational Dermatitis ". The first speaker was Dr. M. W. Goldblatt, Medical Officer, Industrial Hygiene Laboratories, Imperial Chemical Industries Ltd., who spoke on the problems of dermatitis in the chemical industry.

Dr. Goldblatt summarized the changes and development in the chemical industry during the last 30 years. The effect of these had been the almost universal application of chemical methods and products in industries of the most varied kinds. The result was that the industrial worker found himself exposed to a greater extent than ever before to a multiplicity of complex hazards to which his organism had somehow to adapt itself. That such adaptation did occur in the light of the nature of the properties of the elements and compounds used was one of the extraordinary facts of cellular adaptability. Adaptability to environmental factors might seem to follow from evolutionary principles, and so long as these factors fell within the range of so-called natural phenomena the wonder of the process did not strike us too forcibly. But when the environmental factors were manifestly not of the " natural " kind and this applied, *par excellence*, to complex synthetic organic compounds which had no counterpart (at least as far as could be seen at present) in nature, the fact of adaptability was almost unbelievable.

The impact on the skin was immediate and found expression in the so-called direct and indirect irritations. Of the direct irritations there was little more to be said, in general, than that the effects were substantially independent of the individual, that they were understandable on physico-chemical grounds of reaction, oxidation, reduction, lipoid solvency. The normal tolerance to alkalies of any individual skin depended upon the available skin acidity and intolerance resulted from deficiencies of this factor. Sensitivity to alkalies was probably not an immunity reaction in the sense usually assumed. Intimate and repeated contacts with lipoid solvents or detergents, by removing the superficial film of fatty material on the skin, lay bare the unstable stratum corneum with a consequent more ready exposure of the subjacent layers to the insults of the external world and to the industrial products that could establish sensitization.

The mechanism of sensitization dermatitis is obscure. In spite of the large list of industrial materials (especially in the organic field), to which the skin might become sensitized, there were many problems to be resolved before real knowledge could be claimed. For example: What was the precise meaning of contact? To what extent was slight injury required to permit contact sensitization? If the process depended upon antigen-antibody reaction, what was the nature of the antigen? What changes did sensitizing compounds undergo in the skin? What fundamental chemical or physical property was required to render a substance a sensitizer?

Dr. Goldblatt, in reference to the process of spontaneous desensitization, described his own experience of workers engaged in the manufacture of antimalarial compounds. Here the incidence of sensitization dermatitis was notably high and spontaneous desensitization could be demonstrated in a high proportion of patients who had only been allowed to continue at work (during the War when manpower was very short) with minimal mild therapy. The re-establishment of sensitization after a prolonged period of absence from exposure was readily observed. The possible relation of this phenomenon to the negative phase in anaphylaxis was referred to.

Polyvalent sensitivity might present peculiar features. The case was described of a woman research chemist who, in 1923, at the age of 24 years, suffered a severe attack of dermatitis after handling 1.2.4 chlor-dinitro-benzene. After 20 years on library work involving no contact with organic reagents, she returned to laboratory work in 1943, and, in the course of work involving the use of 1.3.4.-chlor-dinitro-benzene (absolutely free from the 1.2.4. isomer), she immediately reacted with an extensive irritant, itching, vesicular eruption which led to great loss of sleep. This case illustrated the inability of the skin to distinguish two close isomers and the maintained sensitivity to a particular kind of chemical compound after many years.

Photographs were shown of a worker who, knowing from experience some ten years previously that he was extremely sensitive to 1.2.4 chlor-dinitro-benzene, and having been given, during the War, a job not involving any contact with this compound, deliberately assured

himself of contact with it and received compensation for a long time for the resulting severe and generalized dermatitis.

Emphasizing the need for every industrial medical officer to take the broadest view of research and not to regard the latter merely as a laboratory activity, Dr. Goldblatt described cases of extensive depigmentation in Negroes arising both in industry (in the U.S.A.) and in private life (in Britain). Both groups of cases offered a rare combination of opportunities for clinical and laboratory work. Both series arose from the intimate contact with the skin of rubber articles of clothing (industrial rubber aprons and gloves and rubber-lined shoes) which contained anti-oxidant compounds which, by inhibiting the dopa oxidase reaction which is an essential step in the formation of melanin, prevents the replacement of the pigment in the pigment layer.

"The Differential Diagnosis of Occupational Dermatitis" was the theme chosen by Dr. R. M. B. MacKenna.

Dr. MacKenna stated that the views of industrial medical officers and dermatologists concerning contact dermatitis tended to differ slightly because, whilst the medical officers saw many cases in which the eruption was due only to primary irritants, and several cases in which resistance to sensitizing agents was rapidly acquired, the dermatological clinics tended to receive as patients many difficult, chronic cases which, because of polyvalent sensitivities, and psychological or other causes, were extremely resistant to treatment. Dermatologists also saw cases in which the diagnosis was in dispute, and therefore he proposed showing a series of coloured lantern slides* of some of the maladies which might be confused with industrial dermatitis.

He then discussed briefly the manifestation on the hands of acrodermatitis perstans acrosclerosis, erysipeloid, erythema multiforme, nummular eczema, lichen planus, lupus erythematosus, self-inflicted injuries, pityriasis rubra pilaris, psoriasis, ringworm, scabies, secondary syphilis and other maladies, comparing these with a few photographs of true contact dermatitis.

In the discussion which followed he said, among other things, that he was convinced that, although it was impossible to estimate the amount of wastage by sickness which had been prevented by the activities of industrial medical officers, he was convinced that if their work ceased suddenly the sickness and accident rates of industrial workers would very rapidly rise.

Dr. Sibyl Horner spoke on "The Prevention of Occupational Skin Conditions".

Dr. Horner stressed the need for measures to prevent the onset of occupational dermatitis and quoted figures of compensated and benefited cases for 1948, pointing out the drain on manpower that such numbers involved.

The substitution for known skin irritants by something more innocuous is the counsel of perfection in the prevention of dermatitis but this is seldom practicable. The selection of workers who will have to be exposed to skin irritants is desirable and a history of eczema or dermatitis is important, whether or not it has occurred during occupation. The presence of skin disease in a candidate for work in which there is a dermatitis hazard does not however necessarily debar him from such employment, for although acne vulgaris would seem to be a condition possibly unsuited for exposure to agents such as mineral oil, pitch, tar and the chlorinated naphthalenes which cause acneiform eruptions on the exposed parts of the skin, such a subject may well thrive in contact with a trichlorethylene degreasing bath, or other degreasing agents, from which the ichthyotic subject is generally best excluded.

A negative patch test is not quite conclusive and positive tests may in some instances be misleading. Protection of workers is important: something more is required than special clothing, and the aim must be essential mechanical protection by satisfactory environmental conditions at work. Barrier creams play a small but important part in the prevention of dermatitis, although recent work of the Industrial Medical Research Unit of the M.R.C. at the Birmingham Accident Hospital has endorsed the view previously held that the efficacy of barrier creams in protecting the skin from contamination from mineral oil is not high. A cleanser composed of 50% soap powder and 48% wood flour is favoured by Dr. Cruickshank. A cleanser composed of 98% neutral sulphonated castor oil with 2% wetting agent, such as alkyl sulphate, has proved of practical value and does not "de-fat" the skin.

Routine inspection of the workers should be carried out in order to detect early signs of skin irritation, and the removal from the skin by harmless methods of traces of skin irritants at the end of a day's work is essential. Reduction of dermatitis calls for the blending of effort of employer, industrial doctor, and employees, while, in the words of a French proverb, "the past and the present blend to make the future".

Following these papers there was a discussion in which Dr. Davidson, Principal Medical Officer of the Ministry of National Insurance, took part.

Dr. Davidson pointed out that none of the speakers had defined what he meant by "occupational dermatitis". To obtain benefit under the Industrial Injuries Act a case of occupational dermatitis must be accepted as falling within the definition "inflammation or ulceration of the skin produced by dust, liquid or vapour or radiant energy". The words "produced by" are not further defined: it might be that a case of dermatitis, not primarily occupational in origin but worsened or rendered incapacitating by conditions of work, had been partly "produced by" the agents stated. Figures for successful claims might, therefore, be misleading.

Dr. Davidson gave examples of what was meant by "loss of faculty" under the Industrial Injuries Act, and drew attention to the mode of assessing disablement resulting from occupational dermatitis. He spoke strongly about the difficulty of arriving at a true diagnosis when expert dermatologists differed, and deprecated the free use by dermatologists of such vague and meaningless phrases as "permanent sensitivity to chemicals" and "permanent sensitivity to paints". Dr. Davidson

*Lent by St. John's Hospital for Diseases of the Skin.

felt also that for dermatologists to offer an opinion on the percentage assessment of disablement when asked for an opinion on diagnosis, and without stating their basis for comparison, was not helpful.

Asked why the Ministry of National Insurance did not notify employers in every case of a successful claim to injury benefit for occupational dermatitis, Dr. Davidson pointed out that this would involve a breach of confidence as well as much added clerical work (perhaps impossible in mid-winter) and that since diagnosis was subject to a series of appeals, the ultimate diagnosis in any given case (although not the benefit payable) might not be available for a very long time.

London Group

Chairman: Dr. M. L. Dobbie-Bateman,
Messrs. Harrods Ltd.,
Knightsbridge, S.W.1.

Hon. Secretary: Dr. R. E. W. Fisher,
Chief Medical Officer,
Metropolitan Division,
South Eastern Gas Board,
589, Old Kent Road, S.E.15.

A meeting of the London Group was held on February 9 at the London School of Hygiene and Tropical Medicine.

A film entitled "Industrial First Aid" made by T. J. Smith & Nephew and directed by Dr. R. A. Trevethick, of Steel, Peech & Tozer, was shown. Dr. Trevethick introduced and explained the film. After the film there was a general discussion in which a number of members took part.

A meeting was held on May 11 at the Luton and Dunstable Hospital and at the Vauxhall Motor Company's works at Luton. At the Luton and Dunstable Hospital Mr. L. W. Plewes, the orthopaedic surgeon in charge of the Rehabilitation Centre explained the method of working of his department. The department comprises, as well as complete and up-to-date physiotherapy arrangements, a workshop where men are treated by being employed on actual industrial work. The work is provided by arrangement with firms in the neighbourhood. The men attend at the Centre either as in-patients or out-patients. Work is so arranged that the movements required are those which are necessary to the patients' recovery. The work is paid for at normal rates by the firms which provide it, but the money, which of course is small because the men are in general capable only of a little work, is retained by the Hospital in a special fund. Mr. Plewes and the engineers in charge of the workshop demonstrated many ingenious devices designed to ensure the correct movements.

At Vauxhall Motors Dr. A. R. Thompson, Medical Officer to Vauxhall Motors Limited, gave a talk on the theory and practice of the Vauxhall re-training shop: this is essentially a shop where the principles in use in the workshop of the Luton and Dunstable Rehabilitation Centre are applied to the work of Vauxhall Motors. Dr. Thompson made it clear that the rehabilitation scheme was dependent upon the close cooperation of the industrial medical officer, the orthopædic surgeon, the re-training shop superintendent, and, last but most important, the patient. After Dr. Thompson's talk lunch was served to the members by courtesy of Vauxhall Motors.

After lunch a film describing the work of the re-training shop was shown. The members then had an opportunity to inspect the work of the re-training shop and found that the principle of rehabilitating men on actual industrial work was being applied with considerable skill and ingenuity. A great deal of informal discussion took place and it was evident that the members were much impressed with what they had seen.

After tea the meeting concluded.

Merseyside Group

Chairman: Dr. E. Holland,
Mersey Docks Medical Services,
Dock Office, Liverpool, 3

Hon. Treasurer: Dr. F. A. Wilson,
Alfred Holt & Co.,
India Buildings, Liverpool, 2

Hon. Secretary: Dr. F. H. Tyrer,
Lever Brothers, Port Sunlight Ltd.,
Port Sunlight, Cheshire.

Mr. Evans gave a talk on prolapsed intervertebral discs. This diagnosis he said was being made with increasing frequency nowadays, and the condition was usually the cause of what was formerly labelled primary sciatica. The original diagnosis of rupture or herniation of an intervertebral disc was made by radiograph, after injection of lipiodol into the spinal theca, and later aerography had been used. Both these methods had now been abandoned, and a typical clinical syndrome was now recognized.

The lesion was most commonly found between L. 4 and 5, or between L. 5 and S. 1. A typical history showed the following features:—

Distribution and Type of Pain.—The pain begins in the lumbo-sacral region, and spreads down the buttock and the posterior or postero-lateral aspect of the thigh into the calf, but rarely into the sole.

Factors Influencing Severity of Pain.—Pain was always aggravated by movement, and relieved by complete rest, especially when the hip and knee of the affected side were flexed and the other leg extended.

Intermittency of Symptoms.—There was frequently a previous history of similar attacks.

Paraesthesiæ.—Usually pins and needles aggravated by the same factors as produced pain.

History of Trauma.—This was given by 50% of cases.

Mechanism of Production of the Lesion.—Typically, this consisted of trauma, produced by attempts to extend the flexed spine against resistance coupled with a rotatory movement of the spine. Spinal flexion

produced slight separation of the posterior margins of the vertebral bodies; contraction of the crura of the diaphragm by respiratory effort then fixed the bodies of L. 1, 2, and 3. The disc bulged below this level, and extension and rotation then nipped the protruding disc.

Physical Signs.—The sex ratio of males to females was 4 : 1, and the average age of incidence 35. There was frequently emotional instability. The spine was quite often rigid, and might be tilted and convex to the painful side. The tension test was positive i.e., pain is produced by flexing the hip with the knee extended when the patient lies supine. There is diminution of tone in the gluteaea and the dorsiflexors of the foot. The knee jerks are normal, and the ankle jerks diminished or absent on the affected side. Radiographs might reveal scoliosis, obliteration of the lumbar lordosis, and diminution of the disc space. The principal conditions to be excluded in the differential diagnosis were metastases, tuberculosis, and spondylisthesis.

Treatment.—The vast majority of cases responded well to rest and physiotherapy. Spontaneous cure was frequent. Economic circumstances might prevent a patient from having a complete rest for a long period, and, for such patients, the head suspension plaster jacket was of great value as it permitted him to return to work while still under treatment. Its effect was to change the relationship of the spine to the disc, and to immobilize the spine. If cure did not occur after three months in plaster, the jacket was replaced by a removable pexaloid jacket, which was worn for 18 months to two years.

Operative Treatment.—This gave good results in a small number of carefully selected cases.

Laminectomy was now seldom performed, the more usual operation being foraminotomy, which consisted in removal of the ligamentum flavum with a small portion of the laminae, exposure and removal of the disc.

Indications for operation were a cauda equina lesion, a recurrent attack after adequate conservative treatment, gross sensory or motor changes of long duration, and sometimes a first severe attack where on economic grounds a rapid cure was necessary.

Mr. Evans stressed that not all heavy work places the nucleus pulposus in jeopardy. Workers often discovered for themselves trick movements which minimized the strain of lifting, e.g., miners with pit trams.

During the rehabilitation period, when patients first returned to work, any weight-lifting done should be from knee level only.

At the conclusion of the talk, a selection of cases was demonstrated, some of whom had been treated by operation, others awaiting operation, and some treated conservatively.

Dr. Douglas Freshwater, consultant dermatologist, demonstrated cases of pityriasis rosea; hæmangioma; button epithelioma; erythema annulare centrifugum; erythema induration; cheiropompholyx due to external irritant; low-grade infective seborrhoeid; acne excoriée de jeune fille; mild occupational dermatitis; accessory auricle; nummular eczema; parakeratosis mibele of penis.

After the demonstration a discussion on the differential diagnosis of industrial dermatitis took place.

The I.C.I. film on industrial dermatitis was shown for the benefit of the members who had not had an opportunity of seeing it at the recent London meeting of the Association.

In the evening a dinner was held in the Bridge Inn, Port Sunlight, at the invitation of Messrs. Lever Brothers. The chairman, Dr. Holland, expressed the thanks of the Group to Dr. Freshwater for his excellent demonstration, and to Messrs. Lever Brothers, Port Sunlight Ltd. for their hospitality.

North Western Group

Chairman: Dr. R. S. F. Schilling,
University of Manchester,
Manchester, 13.

Hon. Secretary: Dr. G. Taylor,
Messrs. A. V. Roe & Co. Ltd.,
Greengate, Middleton, Manchester.

A meeting of the North Western Group was held at Manchester on March 8 when a series of papers was read on radiation medicine.

Dr. E. F. Edson, Principal Medical Officer, Ministry of Supply (Division of Atomic Energy Production), opened the dissussion and pointed out that there were both analogies and differences between the orthodox industrial toxicology and the toxicology of ionizing radiation. Attention was drawn to the importance of establishing permissible concentrations of radio activity in the occupational environment which were in almost every case far lower than those recommended for orthodox non-radioactive toxic hazards in industry.

Dr. Felton dealt with the acute and chronic clinical consequences of external and internal radiation, at dosages and dosage rates vastly in excess of current recommendations for occupational exposure, based on the history of the radium and *x*-ray industries and the military and therapeutic usage of radiation. The changes which took place were protean: blood, skin, gonads, eyes, lungs, bones, gastro-intestinal tract, liver and kidney might all be involved.

Dr. A Butterworth followed with a discussion on possible lines of therapy for the conditions mentioned if by mischance they did arise. Amongst a large number of substances which had been tried experimentally only toluidine blue, protamine sulphate, "rutin", blood transfusion, and antibiotics had any likelihood of frequent success. Theoretically, the pharmacological removal of heavy elements was feasible since their behaviour often simulated that of calcium in the body. The practical value of measures for decalcification was, however, dubious and in general the entire picture of specific therapy for the clinical consequence of excessive irradiation or deposition of radio elements was a depressing one.

Dr. A. S. McLean concentrated upon the hæmatological aspects of over-irradiation with special reference to the diagnostic value of blood counts on radiation workers. The difficulties of assessing the significance of changes noted in the minor degrees of over-exposure was indicated. Dr. McLean thought that the value of blood count procedures in persons exposed to radiation within permissible levels was perhaps more likely to be of psychological value than as a measure of health surveillance. However, this aspect could be reversed if one considered the necessity to avoid employment on radiation work of persons with existing blood disease, or the occasional possibility of over-exposure occurring but not being recorded by the personal monitoring devices, or the technical fallibility of determinations of certain injurious radio elements in the occupational environment.

Dr. T. E. Graham described the possible structure and role of an industrial medical service in radiation industrial establishments, as exemplified by that of the Division of Atomic Energy (Production Factories). The health supervision of radiation risks must not be carried out to the exclusion of more orthodox and accepted needs of employees for the promotion of their occupational health and wellbeing. Pre-employment medical examination was required before employment in the Division of Atomic Energy, and particular attention was paid to the organs or tissues thought most prone to injury by radiation, e.g., bone, bone marrow, blood-forming tissues, lung, kidney, skin, and especially to possible pre-malignant conditions. The examination included a chest radiograph and one or more routine blood counts. Annual clinical examination and chest radiograph followed, and blood counts were carried out every six months or more frequently. A final examination preceded any employee leaving factory employment. The medico-legal aspects were, in all examinations, borne in mind as well as the purely medical ones.

A dental service for the factories was also run, based on the known predisposition of pre-septic or septic dental states to lead to bone changes in the jaw from radio-element deposition.

A chiropody service paid attention to the potential foot trouble arising from the necessary wearing of rubber boots in the many radiation process areas of the factories.

Great importance was attached to the factory casualty service and on injury assessment and injury prevention arrangements. Collaboration between the medical and safety staff was a daily feature of the preventive health measures. Accurate records were maintained of all relevant occupational health data on all employees with the intention of eventual periodic analysis of the data. Information was currently lacking on the progressive health trends in employees exposed for lengthy periods to radiation or radioactive substances. The medical officers found serving on factory committees as observers or advisers of great value. The health and safety services received early opportunities of advising the designers on special points to be considered when a new process or operation had to be converted into plant and building. The medical authorities stipulated the maximal allowable concentrations of permissible exposures for various operations.

Mr. W. Lunning, physicist in charge of the Health and Safety Physics Department at one establishment, dealt with the role of that Department in the interests of the safety and health of the employees. Control of external radiation by "Beta" and "Gamma" radiation was possible either by absorbing the radiation energy in an inert barrier of suitable dense material such as lead, concrete, iron, brickwork, etc. or by increasing the distance between the worker and the source, or by restricting the time permitted for work on the particular operation. Control of internal radiation amounted to controlling the amount of radioactive contamination of the working air and environment with particular attention to dust. The degree of attention to hazard control demanded intensive radiation surveying and atmospheric contamination assessments carried out both as a regular routine and also on suspicion of unusual occurrences. For the measurement of individual exposures, a sensitive photographic film worn for a stipulated period was used. Where the result was required quickly, the issue of a small pocket ionizing chamber was arranged. Atmospheric dusts were collected by filtration or electrostatic precipitation, and could then be assessed by chemical methods or by radiometric counting.

Scottish Group

Chairman: Dr. A. F. Campbell,
Albion Motors Ltd.,
South Street, Glasgow, W.4.

Hon. Secretary: Dr. I. M. Richardson,
Stewarts & Lloyds Ltd.,
Tollcross, Glasgow, E.2.

A meeting was held on April 20 at the Airdrie Factory of Boots Pure Drug Company Ltd. Members were welcomed by Dr. Lloyd Davies and Dr. Hunter, and, after inspecting the health unit, they were conducted round the works.

Later Dr. Lloyd Davies gave an address in which he described his own concept of the future of industrial medicine.

The annual business meeting was held on May 24. The Secretary gave his report and the financial statement. Members expressed dissatisfaction with the present financial policy of the Association preferring a return to the block grant system. The Secretary was asked to report this to the Association treasurer and to have it raised at the next Council meeting.

A letter from Dr. Mekelburg was read requesting possible speakers and subjects for the London meetings. After discussion, the following suggestions were made: Dr. George Buchanan to give his observations on Dupuytren's contracture in dockers; Dr. Swanston to take part in a Symposium on "How is the Industrial Injuries Act Working?".

The last item on the agenda was the syllabus of meetings for 1950-1951. This was duly approved, and after a speech of congratulations from the Chairman to Dr. Meiklejohn on his Bridge Lecture, the proceedings closed.

South Wales and West of England Group

Chairman: Dr. J. Gwynne Morgan,
Chief Medical Officer, Mond Nickel Works,
Clydach, Swansea.

Hon. Secretary: Dr. T. H. Jenkins,
Divisional Medical Officer, National Coal Board,
Institute of Engineers, Park Place, Cardiff.

A general meeting was held in the lecture theatre of the Mining Department, University College, Cardiff, on April 27, 1950, when Dr. J. P. Bull, Assistant Director of the Industrial Medicine Research and Burns Unit, Birmingham Medical Research Council, gave a lecture on Infection with special reference to the hands and feet. After the lecture a sound colour film on "Wound Infection" was shown, followed by a most interesting discussion.

Dr. Bull spoke on the studies of infection made by the M.R.C. Industrial Medicine Research Unit in respect of the engineering industries in the Midlands. He thought the findings might have wide application to other industries.

As regards fungal infections of the feet, limited studies so far had shown that this was less widespread than had been anticipated. Secondary coccal infection was common and might determine exacerbations and require treatment before fungicides could be effective.

Studies of sepsis showed that though it accounted for perhaps less than 2% of factory surgery attendances, it caused between 10 and 30% of lost time accidents and a corresponding proportion of time loss. Of recent industrial fatalities in Birmingham, 6% were attributable to sepsis. In finger pulp and tendon sheath infection a controlled trial of systemic penicillin, combined with careful minimal surgery, had demonstrated its value.

Reference was made to the recent M.R.C. Special Report No. 266 by Williams and Miles. The no-touch technique shown in the film was a logical and practical outcome of these studies. Intelligent layout of dressing stations as described in the April, 1944, issue of the *British Journal of Industrial Medicine* was a further help. Infection between dressings could readily occur when a "dry dressing" became soaked. Waterproof and inpermeable dressings were a partial answer, but had disadvantages. Recent experiments with a nylon film dressing, which permitted the wound to be seen and had other advantages over the usual adhesive dressings, were promising.

Members asked Dr. Bull a number of questions, and an interesting discussion ensued.

Tees-side Group

Chairman: Dr. W. Gillies Annan,
41, Cleveland Road,
Darlington, Co. Durham.

Hon. Secretary: Dr. J. B. Adamson,
Ministry of Fuel and Power,
Crown Buildings,
63, West Gate Road,
Newcastle on Tyne, 1.

A meeting was held on May 11, 1950, at Stockton and Thornaby Hospital.

Eight members and four visitors attended the meeting, and after the business part of the meeting two films, produced by the film unit of Imperial Chemical Industries Ltd., were shown.

The first film, entitled "Birth of a Drug", showed the various steps by which a new drug comes into being: discussions between chemists on various chemical formulæ; the making of various substances with trials on animals; the finding of a substance suitable for trial on human beings; cooperation of a physician in testing the drug; and finally, when it has been proved to be of value in the treatment of a disease, the manufacture of the drug on a commercial scale.

The second film, entitled "Industrial Dermatitis", began with a description of the skin and went on to show how the various hazards in industry could disturb the proper functioning of the skin and so lead to dermatitis. Examples of industrial dermatitis were shown and steps to be taken to avoid it discussed.

Yorkshire Group

Chairman: Dr. C. Cresdee,
I.C.I. Ltd. (Dyestuffs Division),
Huddersfield.

Hon. Secretary: Dr. B. P. R. Hartley,
Samuel Fox & Co. Ltd.,
Stocksbridge Works, Nr. Sheffield.

The group met on February 16, 1950, at Scunthorpe, Lincs. where they visited selected departments of the Appleby Frodingham Steel Company.

First visited was the south melting shop, the most modern in the Empire, comprising two 380-ton Siemens open hearth furnaces of the tilting type and a mixer. Members were impressed with the spaciousness of the layout and evidence of good housekeeping. Subsequently the new section mill was visited, and members saw the rolling down of steel ingots into smaller sections. The chief impression gained in this completely mechanized mill was of the few personnel who seemed to be required to operate it. The main hazards of the heavy steel industry are accidents and the long term effects of exposure to high temperatures in a dusty atmosphere. In both shops visited these hazards were reduced to a minimum by good working conditions.

Later in the medical department Dr. F. A. Shackleton

showed a case of scleroderma, and Dr. O. P. Llewellyn reviewed a selection of chest radiographs showing all stages of silicosis and other chest disorders.

A meeting was held on May 11 at Salts Mill, Saltaire, Shipley. This worsted mill deals with all processes "from the sheep's back" to the finished cloth. The founder of the firm was a pioneer in many ways. He built his mill in sylvan surroundings and away from the depressing conditions in Bradford, and a model village, the first of its kind, for his employees. The mill was established in 1853, but has been extensively modernized.

Members were shown all processes from sorting the wool to the finished cloth, including the complicated machinery for combing the wool and spinning.

The medical officer, Dr. W. Ward Smith, showed members the medical facilities which included the services of a visiting ophthalmic surgeon, dental surgeon and chiropodist, for whom adequate accommodation and equipment were provided.

Woollen mills do not have the condition of high humidity and temperature found in cotton spinning mills, and in this case the working environment, though warm, was not uncomfortable despite the fact that it was a hot day. The hazards of the industry are those associated with moving machinery, and contact dermatitis due to alkalies and other processing solutions used. In the mill visited the incidence of accidents and dermatitis was very low.

ABSTRACTS

(This section of the JOURNAL *is published in collaboration with the two abstracting Journals,* Abstracts of World Medicine, *and* Abstracts of World Surgery, Obstetrics, and Gynaecology, *published by the British Medical Association. The abstracts are divided into the following sections: toxicology; industrial physiology; industrial lung disease; industrial skin diseases; accidents and orthopædic surgery; industrial ophthalmology; environment; general. Not all sections will necessarily be represented in any one issue)*

TOXICOLOGY

Deficiency Patterns in Metallic Poisoning. HUGHES, W. (1950). *Brit. med. J.*, **1**, 634.

The hypothesis which led to the discovery of 2 : 3-dimercaptopropanol (dimercaprol, BAL) was that lewisite poisoned the pyruvate-oxidase system by its avidity for the SH groups of the protein constituent of the enzyme. In many enzyme systems, however, the co-enzymes contain members of the vitamin-B complex, while, in addition, vitamins and factors other than the B complex may form linkages with SH–containing proteins with which a heavy metal may interfere in the same way. Thus, as a result of "biochemical lesions", reactions associated with specific vitamin deficiencies may occur in cases of metallic poisoning.

Two cases of gold poisoning are described in which the pattern of lesions was that which might be expected in vitamin-K and in vitamin-B_2 deficiency respectively. The first patient was a man of 61 with a 20-year history of rheumatoid arthritis, in whom treatment with sodium aurothiomalate (50 mg. weekly) led to a severe epistaxis and a petechial rash. Complete recovery followed the injection of 2 ml. twice daily of a 10% solution of dimercaprol to a total of 28 ml. The second was a woman of 52 with advanced rheumatoid arthritis of 29 years' duration, recently treated with sodium aurothiomalate. This resulted in a rash which included an extensive area of the perineum and buttocks. Although the daily administration of 20 mg. of riboflavin by injection and 30 mg. orally did not influence progress, 26 injections of dimercaprol (2 ml. 10% solution a day) resulted in complete recovery. In a case of exfoliative dermatitis, in which a generalized erythema with desquamation in and on the axillæ, groins, elbows, and dorsa of the hands and feet developed 4 days after the end of treatment for secondary syphilis with neoarsphenamine and bismuth, the condition was rapidly and successfully treated with 2-ml. injections of 10% dimercaprol daily for 7 days. A fourth case is described in which a man of 75 was treated for gross cardiac œdema with 7 injections of 2 ml. of mersalyl on alternate days, after which a morbilliform rash developed on the face, arms, and legs, together with conjunctivitis and small buccal ulcers. The rash became generalized, but gradually cleared after the injection of 2 ml. of 10% dimercaprol twice daily for 3 days.

The author suggests that the "biochemical-lesion" hypothesis should be kept in mind in cases of "pellagra sine pellagra" and other epithelial patterns of deficiency unassociated with any obvious malnutrition, which might possibly be due to metallic poisoning. But the view put forward by Cohen that the success of dimercaprol in metallic poisoning cases will enable higher doses of gold to be used in the treatment of chronic rheumatism is accepted only with reservations, cases having already been reported in which treatment of gold intoxication with dimercaprol led to relapse in the rheumatic condition. *Malcolm Woodbine.*

Ferrous Sulphate Poisoning. Its Incidence, Symptomatology, Treatment, and Prevention. THOMSON, J. (1950). *Brit. med. J.*, **1**, 645.

The author reviews the recent literature from English sources on poisoning with ferrous sulphate in the form of tab. ferri sulph, co. *B.P.C.*, and incorporates the details of 6 cases of accidental poisoning in children varying in age from 11 months to 4½ years studied at the Royal Infirmary, Dundee. Three of the cases have already been reported (Thomson, *Brit. med. J.*, 1947, **1**, 640, and Prain, *ibid.*, 1949, **2**, 1019), and the details of the new cases added are similar in all respects.

Two fatalities occurred, both in children of approximately 18 months, one of whom had swallowed 39 tablets (each containing 3 grains (0·2 g.) of ferrous sulphate, with copper and magnesium sulphates, $^1/_{25}$ grain (2·6 mg.) of each) and the other 10 : the remainder, who recovered after treatment, had taken smaller doses, reduced in all except one case by vomiting. The outstanding features were pallor, drowsiness, vomiting (often blood-stained), and melæna. A tendency to relapse in spite of generous lavage with sodium bicarbonate solution and administration of bismuth carbonate or magnesium hydroxide was noted, and BAL which was given in doses of 0·5 ml. 4-hourly in one case for 24 hours, did not appear to have any value. Necropsy findings in the 2 fatal cases included inflammation of the gastric mucosa with bleeding from necrosis and erosions, and similar changes of less intensity in the small bowel.

The author describes the warning on the container of commercial brands of these tablets not to leave them within reach of young children, "who may eat them as

sweets with harmful result" as inadequate for the prevention of a serious form of poisoning.

Keith Simpson.

Disodium Hydrogen Phosphate Therapy in Lead Poisoning. PROCTOR, C. D. and KAHN, H. S. (1950). *Amer. J. med. Sci.*, **219**, 316.

In 8 patients, in whom there were physical signs, symptoms, or histories pointing to lead poisoning, lead excretion in the urine was studied; in 3 the amount excreted was 450 μg. or more, in 4 between 150 and 200 μg., and in 1 below 150 μg. per litre. All amounts except the last would be classified by Gant as "definitely toxic". Treatment with disodium hydrogen phosphate was established, 2 patients in the first group being given 60 gr. (4 g.) by mouth thrice daily for 15 days and the third 10 gr. (0·65 g.) intravenously thrice daily for 7 days. Two of the second group were given 60 and 30 gr. (4 and 2 g.) by mouth thrice daily for 5 days, the other two 5 gr. (0·32 g.) intravenously, thrice daily, also for 5 days. The last was given 15 gr. (1 g.) thrice daily by mouth for 7 days. In all cases there was complete remission of symptoms soon after treatment started, and no abnormal substances appeared in the urine. After the close of the treatment lead values were again determined in the urine; the figures showed a reduction in most cases to one-third or one-half of the previous figures. In 3 cases determinations were also made during treatment and showed a considerable rise in the level, in one nearly to five times the original figure.

Three control cases were also investigated, in which the levels of lead in the urine before treatment were 87·2, 17·5, and 31 μg. per l. respectively. Disodium hydrogen phosphate, 15 gr. (1 g.) by mouth thrice daily, was taken for 5 days. During the treatment the urinary content of lead rose to 152·3, 48·9, and 135·6 μg. per l. respectively, but on the fifth day after the end of treatment the figures were 38·2, 10·1, and 25 μg. respectively. No symptoms were observed and no abnormal substances found in the urine. It is claimed that the drug is non-toxic, and causes considerable amounts of lead to be removed from the body without the occurrence of any symptoms of acute exacerbation of lead poisoning.

Reginald St. A. Heathcote.

Haemachromatosis. DAVID, G., and SÜMEGI, I. (1950). *Orv. Hetil.*, **91**, 162.

The authors describe a case of hæmochromatosis in a man of 35, on whom they carried out a wide variety of investigations; the more significant findings were the nature of the skin biopsy specimens, the diabetic glucose-tolerance curve, the evidence of hypogonadism, and the finding of large amounts of coproporphyrins in the urine, mainly of type I but with some of type III. The authors discuss the various theories of the ætiology of the disorder; they consider it to be a constitutional disturbance of mineral storage, analogous to the carbohydrate-storage disturbance in Gaucher's disease and to the protein-storage disturbance in the dysproteinæmia described by Wuhrmann and Wunderly. The importance of the porphyrinuria is discussed in part II of their paper.

Dushanka Wolstenholme.

Haemochromatosis. The Pathogenesis of Porphyrinuria in Lead Poisoning and Haemachromatosis. SÜMEGI, I. (1950). *Orv. Hetil.*, **91**, 165.

The author carried out experiments to confirm his opinion that both in hæmochromatosis and lead poisoning the iron and the lead respectively cause generalized damage to the reticulo-endothelial system, the extent and principal site of the damage being reliably indicated by the presence and type of coproporphyrin in the urine.

When white rats are given lead acetate daily in a 1% solution intragastrically, in a dose of 1 ml. per 100 g. body weight, porphyrinuria is found at the end of the first week, although fluorescence in the bone marrow is detectable microscopically only; cultures of the marrow from these animals show much fluorescence within 48 hours. The author believes that when this porphyrin is formed in the marrow iron is no longer taken up in the formation of hæmoglobin and that the substance which is formed without iron is coproporphyrin III; the anæmia in lead poisoning is partly due to this failure to take up iron and partly due to the metallic damage to the reticulo-endothelial cells which prevents them from releasing iron normally for blood formation.

When the reticulo-endothelial cells of white rats are thoroughly blocked by Indian ink before administration of the lead as above, it is found that the animals show no evidence of porphyrin formation at the end of one week and only one-third of them have a minimal production after 2 weeks; anæmia develops in these animals well before its appearance in the animals given lead only.

The author believes that in hæmochromatosis the deposition of iron in the liver damages the parenchymatous cells and leads to the appearance in the urine of coproporphyrin I; because of the liver's power of regeneration this appearance is inconstant in the early stages of the disease, which explains the variable findings of other authors. When the deposition of iron is such as to affect seriously hæmoglobin formation in the marrow, then coproporphyrin III is formed. The appearance of type I or III in hæmochromatosis and heavy metal poisoning depends on whether the damage is predominantly of the liver or the marrow.

Dushanka Wolstenholme.

Poisoning with Petroleum Products and Benzol in Children. SCHOLZ, R. (1950). *Klin. Med. Wien.*, **5**, 78.

The symptomatology of poisoning with petrol, benzine (a petroleum derivative), and benzol (benzene, a coal-tar product) is reviewed and the effects on 10 children of ingestion of these substances is described. Seven children, aged 14 months to 5 years, drank varying amounts of petrol. The symptoms observed were: tachycardia, respiratory distress with greatly increased rate of respiration, pyrexia, restlessness, and vomiting. The smell of petrol could be detected in the fæces for many days. The mucous membranes of the mouth were inflamed, but this subsided rapidly. Injury to the mucous lining of the digestive tract was more severe. In the case of another child (aged 14 months) petrol had been accidentally poured over its left shoulder; this caused a

weeping dermatitis in the exposed area with fever and subsequent vomiting, tachycardia, and rapid respiration.

The other 2 children swallowed patent stain removers containing benzine and benzol. Owing to the great affinity of the latter substance for tissue lipids it is more rapidly toxic than the petroleum products. In these cases the central nervous system was affected, with increasing giddiness ending in complete loss of consciousness. The children were comatose and pale, with poor reflexes. The mucous membranes of the mouth were red and inflamed. In one case there was a severe leucocytosis with toxic degeneration of the neutrophils.

In none of the 10 cases were the kidney or bladder damaged, and all 10 patients recovered. The treatment consisted of rapid gastric lavage, saline purgation, and administration of charcoal and circulatory stimulants. In the cases with loss of consciousness, analeptics such as nikethamide were given with apparent benefit.

V. N. Warren.

A Recovery from Ethylene Glycol (Anti-Freeze) Intoxication. A Case of Survival and Two Fatalities from Ethylene Glycol Including Autopsy Findings. KAHN, H. S. (1950). *Ann. intern. Med.*, **32**, 284.

Three stages in ethylene glycol intoxication, usually due to drinking anti-freeze mixture, are described. The first stage, or cerebral stage, is manifested by symptoms resembling those of acute alcoholic intoxication and usually ends in coma. It is associated with cerebral œdema. The second stage is manifested by pulmonary œdema. The third appears later, if the patient survives the first two, and is associated clinically with symptoms and signs of acute nephritis and uræmia. Curiously enough, this stage seems to be associated with the histological findings of a calcium oxalate nephrosis. It usually ends fatally, but in one of the 3 cases described in this paper the patient had completely recovered 5 weeks after drinking 240 ml. of anti-freeze solution (minimal lethal dose 100 ml.). His recovery is attributed to his youth and physical stamina and to the fact that treatment with oxygen and supportive measures was started within 3 hours.

S. S. B. Gilder.

Fluoroacetate Poisoning. A Review and Report of a Case. GAJDUSEK, D. C., and LUTHER, G. (1950). *Amer. J. Dis. Child.*, **79**, 310.

Sodium fluoroacetate was introduced during the war as an efficient rodenticide, but unfortunately its action is not selective and it is highly toxic to man. The authors report the case of a 2-year-old Negro boy who licked the cap of a bottle containing this rat poison. Persistent vomiting developed, and 6 hours later convulsions and stupor. On admission he was in tetanic spasm, comatose, and with irregularity of the heart and respiration. Intravenous administration of calcium gluconate relieved the tetany and the child became quite flaccid. For the next few hours the depth of the coma varied, and he had one convulsion. Next day the child was still limp and unresponsive, but had frequent tonic spasms. He did not respond to painful stimuli. The heart remained irregular, and there were periods of fibrillation and of paroxysmal tachycardia. On the 3rd day the tonic convulsions gradually subsided, but the child remain comatose. The pulse was regular but he had two periods of apnœa requiring artificial respiration. On the evening of the 4th day—100 hours after taking the poison—the child became conscious and by the 6th day he was clinically well. He was discharged well on the 11th day. Chemical analysis proved that the substance in question was a solution of sodium fluoroacetate, and that it contained 0·135% of fluorine. The clinical picture closely parallelled that seen in experimental poisoning in laboratory animals. No antidote is available for the poisoning, but sedation with barbiturates offers a possible means of combating the convulsions.

Gilbert Forbes.

Fatality and illness Caused by Ethylene Chlorhydrin in an Agricultural Occupation. BUSH, A. F., ABRAMS, H. K., and BROWN, H. V. (1949). *J. industr. Hyg.*, **31**, 352.

Seed potatoes are treated with ethylene chlorhydrin to hasten their germination. The substance has caused the death of one man and illness of 5 others in California. The death occurred in a man of 34. He complained of nausea, dizziness, vomiting, abdominal pain, weakness, and impaired vision. He collapsed, became comatose, and died 8 hours after the onset of symptoms. On examination, he was deeply cyanosed, the skin was cold and clammy and the blood pressure unobtainable. The urine contained a cloud of albumin. Necropsy revealed severe fatty infiltration of the liver, marked œdema of the brain, passive congestion of the lungs, dilatation of the right side of the heart, passive congestion of the spleen, cloudy swelling of the kidneys, petechiæ of skin and right conjunctivæ, slight ascites and hydrothorax, and fatty degeneration of myocardium. The five survivors had similar symptoms as well as a burning sensation in the nose and irritation of the eyes, and numbness of hands and fingers. Four were admitted to hospital. Two had a marked fall in blood pressure. In one case symptoms persisted for 76 days. The others recovered in hours after the exposure.

Ethylene chlorhydrin is a potent poison affecting nervous, hepatic, renal, and vascular systems. It is absorbed readily through the skin, intestine, and respiratory tract. The maximum allowable concentration in the atmosphere should be 2 parts per million and no contact with the skin should be permitted.

Mechanical ventilation should be used in all chambers where potatoes are treated and 500 continuous air changes are necessary before a chamber is opened. The ventilation should continue while the chamber contents are unloaded.

K. M. A. Perry.

The Toxicity of Ozone. A Report and Bibliography. THORP, C. E. (1950). *Industr. Med.*, **19**, 49.

This paper presents data on ozone toxicity based on experiments and experience of Armour Research Foundation staff members, supplemented by a critical evaluation of the published work of other investigators. It is shown that much of the early work on this subject

was with ozone in air inadvertently contaminated with nitrogen oxides. Inasmuch as the toxic limits of mixtures of these two substances in air are distinctly lower (1·0 part per million) than for either pure nitrogen oxides (20 parts per million by weight) or pure ozone alone (20 parts per million by weight), it is easily understood why ozone was considered by many to be a highly toxic substance. Later workers, taking cognizance of the synergistic effect of nitrogen oxides, found ozone tolerance by humans to be far higher than previously suspected, even as high as 50 parts per million.

A graph is presented showing ozone concentrations and duration of exposure required to produce various physiological effects classified as: non-symptomatic, non-toxic symptomatic, non-toxic irritant, temporary toxicity, permanent toxicity, and fatal. The limits of permanent toxicity and fatal zones are only estimates, inasmuch as the study of physiological effects with these very high concentrations (1 to 30 weight per cent.) has not been undertaken. The absence of reported fatalities is attributed to the fact that heretofore concentrations of potentially lethal order have not been encountered on any appreciable scale. It is evident that lower concentrations of pure ozone of the order of 50 parts per million have no cumulative effect. Treatment based on that recommended for other gaseous oxides is described for ministering to subjects exposed in the permanent toxic range.

Precautions in handling liquid ozone of various concentrations in liquid oxygen are outlined, and means for preventing the accumulation of high gas concentrations during evaporation of liquid are described.—(Author's summary.)

Ophthalmoneurologic Changes in Carbon Monoxide and Some Other Forms of Poisoning. ALMGREN, S. (1949). *Nord. Med.*, **42**, 1898.

Interest has been taken in the ophthalmological signs and symptoms produced by carbon monoxide poisoning since the publication by Kugelberg in 1945 of material taken from bus workers in Stockholm. The symptoms may be subjective, or definite ophthalmological signs may be found. The latter are disturbances of fusion and convergence. Kugelberg found these disturbances present in 45% of cases. In 197 cases with ophthalmological findings at the author's clinic 78 had convergence alteration, 69 convergence and fusion disturbance, and 50 fusion disturbance only. The detailed findings are tabulated and summarized. Follow up of 66 cases showed that the time taken for disappearance of the signs on ophthalmological examination varied between 6 months and 2½ years, but in some cases the signs persisted for longer than this.

In addition to carbon monoxide other toxic substances such as trichlorethylene, benzene, propane, and olefines, may cause similar disturbances in persons exposed to their fumes and under conditions of poor ventilation.

Kenneth Bowes.

Arsine Poisoning in Industry. Report of a Case. STEEL, M., and FELTHAM, D. V. G. (1950). *Lancet*, **1**, 168.

A Study of Workers Exposed to Talc and Other Dusting Compounds in the Rubber Industry. HOGUE, W. L., and MALLETTE, F. S. (1949). *J. industr. Hyg.*, **31**, 359.

Talc is a hydrous magnesium silicate with the formula $Mg_3Si_4O_{10}(OH)_2$. Similar minerals are pyrophyllite $Ca_2Mg_5Si_8(OH)_2$, serpentine $M_3Si_2O_1(OH)_2$, and dolomite $CaMg(CO_3)_2$. Tremolite as found in nature with talc has a fibrous variety which is used to some extent as asbestos. Soapstone is the granular to cryptocrystalline form of talc. Pneumoconiosis has been shown to be produced by tremolite and pyrophyllite. In the present investigation, 20 men exposed to talc as defined above were investigated by physical and radiological examination. They had worked for periods between 10 and 36 years making the rubber inner tubes of tyres which are dusted with this talc. No abnormality was found. Only one out of 20 men in another group who had worked between 10 and 25 years exposed to whiting (calcium carbonate), pyrophyllite, and talc showed evidence of pneumoconiosis. This man had been exposed to 150 million particles of dust per cubic foot, which was the highest exposure of them all, but he had been a coal miner for 5 years. The vital capacity of all the workers was good. The authors considered that this study showed that long exposure to talc does not produce pathological lesions in the lungs, and that therefore proposals that limits for silicate minerals be added to lists of maximum allowable concentrations should include careful definition of chemical and mineralogical composition.

K. M. A. Perry.

INDUSTRIAL PHYSIOLOGY

Pulmonary Function Studies in Bituminous Coal Miners. MOTLEY, H. L. (1950). *W. Va med. J.*, **46**, 8.

Physiological tests were carried out on 56 "anthracosilicotic" bituminous coal miners, with an average period of underground exposure of 32 years, with the object of assessing respiratory disability. It is suggested that in order to assess respiratory disability the essential measurements are: (1) Ventilation: vital capacity and maximal breathing capacity; (2) degree of clinical emphysema (as estimated from the volume of residual air in relation to total lung volume); (3) ventilation efficiency (as indicated by O_2 uptake and CO_2 output); (4) arterial-blood gas content, especially the percentage O_2 saturation at rest and on exercise; (5) in some cases, pulmonary circulation measurements, including pulmonary arterial pressure and pulmonary vascular-bed resistance.

In the group of subjects studied no relation was found between the degree of clinical emphysema present and the stage of silicosis, judged radiologically (it being considered that if the maximal breathing capacity is less than 40 litres per minute, there is a significant degree of emphysema, and if more than 100 l. per minute no significant emphysema, intermediate figures being of little value by themselves). It was found that in cases of chronic pulmonary disease there was a rise in pulmonary arterial pressure and pulmonary vascular resistance during exercise, the normal finding being reversed. A

distribution difficulty is considered to be the main factor hindering O_2 transfer between alveolus and arterial blood in cases of anthracosilicosis; in some alveoli, fibrosis of the walls obliterates the capillaries, while in other areas it impairs air circulation; in either case venous blood is returned to the arterial circulation. This "distribution factor" is unrelated to the clinical or radiological degree of emphysema. It was found that single estimations of arterial-blood O_2 saturation with the subject at rest were of limited value compared with similar estimations during exercise, inability to sustain or increase the percentage O_2 saturation during exercise indicating respiratory disability. Good correlation was observed between the results obtained in these miners and those in a previously studied group of anthracite miners. *L. W. Hale.*

Penetration of Air-borne Particulates Through the Human Nose. II. LANDAHL, H. D., and TRACEWELL, T. (1949). *J. industr. Hyg.*, **31**, 55.

This paper describes further experiments designed to measure the percentage penetration of a particulate dust or fog of various substances through the human nose, in continuation of work previously reported (Landall and Black, *J. industr. Hyg.*, 1947, **29**, 269). The materials used were methylene blue, bismuth subcarbonate, glycerol, sodium sulphate, and tyrosine, the material being dusted or atomized continuously into a current of air. Samples of this fog before and after passing through the nose were drawn into cascade impactors which fractionated the material into ranges of particle size. Five subjects were used and it was estimated that the standard deviation for penetration in the range of 25 to 75% was 8 or 9%.

The authors describe a new measure of impactibility (as measured in the cascade impactor)—the equivalent impaction diameter—which is defined as "the diameter equal to that of a spherical particle of unit density which impacts in the same manner as does the particle or aggregate in question". When this was used as the variable against which to plot the percentage penetration of the nose by clouds of varying particle-size, the points for all eight materials lay approximately along the same curve, showing that the percentage of small particulate matter penetrating the nose varies inversely with the equivalent impaction diameter and that impaction is the chief factor in removal of air-borne particles from air passing through the nose. It was also shown on one subject that nasal hair plays a significant role in removing particles, the percentage penetration being notably higher after removal of most of the hairs from one nostril. *R. S. F. Schilling.*

Experimental Observations on the Effects of a High Rate of Climb on the Human Organism. EVRARD, E. (1949). *Brux.-méd.*, **29**, 3629 and 3689.

The effect on man of decompression to a simulated altitude of 33,000 feet (10,000 m.) at an average rate of 143 feet (44 m.) per second has been examined. Observations were made in particular on (1) the effect of gaseous dilatation, and (2) the effect of air embolism. The effect was noted of gaseous dilatation on the middle ear, sinuses, alimentary tract, and respiratory system. Figures are given for vital capacity, and alveolar-air composition. The effect on the cardiovascular system was examined from the point of view of pulse rate, blood pressure, vasomotor changes, electrocardiography, blood count, and urinary output. The effect of precipitation of dissolved gases is dealt with relatively briefly.

With regard to subjective and psychic symptoms, a mental excitability followed or accompanied by lethargy was constantly observed in the three subjects after decompression runs. It is suggested that the degree of psychic tension may be a contributory factor in the production of air embolism. The relative merits of pressurization at 2 and 3 lb. per sq. in. (0·14 and 0·21 kg. per sq. cm.) are discussed. *T. C. D. Whiteside.*

Solubility of Lead Compounds in Human Pleural Fluid and Blood Serum. HARROLD, G. C. (1949). *J. industr. Hyg.*, **31**, 327.

Since workers exposed to lead chromate do not appear to suffer ill effects, the solubility of various lead compounds was investigated. It was found that the solubilities of lead chromate and titanate were of a lower order than those of lead oxide and carbonate. The solubilities of lead oxide and carbonate in human lung fluid and blood serum were comparable to that in sera of other animals. The solubility of impure lead titanate in triple distilled water is over twice the solubility of purified lead titanate, whereas impure lead chromate is more soluble in distilled water because of the soluble chromate in impurities. Hence the solubility of the purified insoluble lead salts in body fluids will differ from that of impure commercial products, owing to the much greater solubility of the impurities in lung fluid and blood serum. The data lead to the conclusion that purified lead titanate is less soluble than the impure salt. The impurities in lead chromate are probably in the form of free chromate ion and hence do not affect the solubility of the lead salt in lung fluids, except that this ion acts as a common ion to reduce the solubility. *K. M. A. Perry.*

Effects of Bed Rest on Cardiovascular Function and Work Performance. TAYLOR, H. L., HENSCHEL, A., BROŽEK, J., and KEYS, A. (1949). *J. appl. Physiol.*, **2**, 223.

Six healthy young men whose ages were from 20 to 33 years were studied before, during and after a 3- to 4-week period of bed rest. Measurements were made under rigidly controlled conditions on the performance of the cardiovascular system (at rest, in the upright posture, and during work), and on speed, coordination, and strength. A high degree of conditioning for the performance of all tests was achieved before the bed rest period.

Bed rest produced a 17% decrease in heart volume and an 8% decrease in the transverse diameter of the heart. There was a highly significant increase in the resting pulse rate, which averaged roughly 0·5 beats per minute for each day of bed rest. The pulse rate at the end of a half-hour walk at 3·5 miles per hour and 10% grade

increased by 40 beats per minute after bed rest. There was no change in the mechanical efficiency during this walk, as the result of bed rest. The oxygen intake during a 90-second run at 7 miles (11·26 km.) per hour and 15% grade was reduced by 730 cc. of oxygen or 16% after 3 to 4 weeks of bed rest. This was accompanied by increases in oxygen debt and blood lactate after the run and a decrease in mechanical efficiency. The maximal oxygen intake was determined in 2 men who showed decreases of 13 and 22% after bed rest. Bed rest produced a marked deterioration in the cardiovascular response to posture as measured by pulse rate and blood pressure changes produced by tilting to 68° on a tilt table. Ataxiameter studies showed that a definite increase in sway resulted from bed rest. Coordination, as measured by pattern tracing, suffered a small loss as the result of bed rest while speed of small hand movements, of medium arm and hand movements, and of gross body and arm movements showed no deterioration. Grip strength was not influenced by bed rest and back strength showed only a small deterioration.

After bed rest the rate of recovery of the various functions was roughly proportional to the extent of the deterioration in bed rest. Strength, coordination, and postural sway recovered early (4 days); blood lactate after exhausting work, and the oxygen cost of exhausting work recovered at an intermediate time (2 weeks); pulse rate during grade walking and O_2 intake during exhausting work recovered late (between 2 and 5 weeks), and the cardiovascular response to posture was very late in returning to normal (after 7 weeks). In one man the effect on the principal components of fitness of a herniorrhaphy with bed rest for 3 weeks was of the same order of magnitude as bed rest alone.

It is concluded that the deconditioning due to bed rest has special characteristics, with the major loss of performance occurring in the cardiovascular system.—(Authors' summary.)

Hemoglobin Concentration and Physical Fitness. CULLUMBINE, H. (1949). *J. appl. Physiol.*, **2**, 274.

The coefficients of correlation between the blood hemoglobin concentration and the responses to various exercise tests have been calculated for Ceylonese subjects aged 10 to 20 years. It is concluded that the blood hemoglobin concentration is correlated significantly with speed of movement, strength and the ability to sustain prolonged moderate muscular effort. There is no evidence for a significant correlation between the hemoglobin level and either the response to moderate exercise or the response to severe exercise.—(Author's summary.)

Relationship between Resting Pulse Rate, Blood Pressure, and Physical Fitness. CULLUMBINE, H. (1949). *J. appl. Physiol.*, **2**, 278.

One thousand Ceylonese subjects, aged 10 to 25 years and of both sexes, performed a series of exercise tests. It has been concluded that the slower the resting pulse rate or the lower the resting systolic blood pressure, the slower is the post-exercise pulse rate or the lower is the post-exercise systolic blood pressure, respectively. Speed of movement, strength and the ability to sustain moderate exercise are positively correlated with the resting systolic blood pressure.—(Author's summary.)

INDUSTRIAL LUNG DISEASES

Reaction to Carbon Dioxide in Pneumokoniosis of Coalminers. DONALD, K. W. (1949). *Clin. Sci.*, **8**, 45.

In view of the doubt about the part played by emphysema in causing disability in pneumoconiosis and the finding of impaired response to inhalation of 4% carbon dioxide in the former condition the authors studied the response of 56 coal miners with pneumoconiosis to such inhalation. Tracings in 8 cases were unsatisfactory and were rejected.

Patients were placed in five groups according to the severity of dyspnœa. In group V, with dyspnœa at rest, 3 out of 4 showed impairment of response to 4% CO_2; 2 out of the 3 had no radiological evidence of pneumcconiosis but only of severe emphysema. In group IV, with dyspnœa on slight exertion, 6 out of 11 gave an impaired response and 2 of these had minimal signs of pneumoconiosis though all 6 had evidence of emphysema. Of the 5 with a normal response 4 had massive shadows in the radiograph. In group III only 3 out of 10, and in group II only 1 out of 19, had an impaired response, while all 4 with minimal dyspnœa (group I) gave normal reactions.

In 5 cases in which there was little evidence of pneumoconiosis but severe emphysema, response to CO_2 was very poor; in 11 out of the 13 with a poor response there was clinical and radiological evidence of emphysema, whereas in only 6 out of 34 with a normal response was there any such evidence. It is thus clear that the response is impaired only if true emphysema is present. It is also clear that severe dyspnœa may in this disease be associated with absence of emphysema and that the dyspnœa is in many cases not due to emphysema. Where emphysema is absent the degree of disability is not related to the severity of the pneumoconiotic lesions.

S. S. B. Gilder.

A Case of Pulmonary Berylliosis, with Pathological and Biochemical Data. POLICARD, A., REY, —, ROCHE, L., and SIMEON, —. (1950) *J. franç. Méd. Chir. thorac.*, **4**, 39.

The patient whose case is described was engaged in the manufacture of alloys and was exposed to dust containing beryllium oxide while cleaning the apparatus. This exposure continued for about 3 weeks only. One to 2 weeks after the cessation of exposure he began to experience slight breathlessness and cough, which increased steadily. About a week later a radiograph showed small nodular lesions in the lower fields of both lungs and increased bronchovascular markings in the same area. Shortly afterwards he was admitted to hospital with greatly increased dyspnœa and cough. He was cyanosed, his temperature was 39° to 40° C. (102·2° to 104° F.) and fine rales were heard throughout both lungs. No tubercle bacilli were found in the

sputum on repeated examination. A second radiograph taken a week after the first showed extensive lesions, suggesting bronchopneumonia, throughout most of both lungs, though the extreme apices and bases were spared. There were confluent nodular shadows in the same areas. The heart appeared normal. The patient became worse, the condition not responding to oxygen, penicillin, or streptomycin administration, and he died just over a month after the last exposure to beryllium. At necropsy very few alveoli were seen to be aerated. There were numerous irregular areas suggesting anthracosis throughout both lungs. There was no evidence of emphysema. The bronchial lymph nodes were enlarged and resembled those in anthracosis, though very soft in consistency. Microscopically, there were numerous bands of young fibrous tissue running through the lung. Many of the alveoli were filled with fibroblasts, lymphocytes, plasma cells, and large monocytic histiocytes; there was relatively little collagen. In the bronchi the elastic tissue was disrupted and the muscle had often disappeared. Many of the smaller bronchioles had disappeared altogether in the masses of carnified tissue. Most of the normal lung capillaries had been obliterated and the pulmonary arteries showed endarteritis with proliferation of the intima. There were no giant cells, no specific granulomatous tissue, and no necrosis. The "anthracotic" areas consisted of masses of monocytes stuffed with black spherical granules 1 to 2 μ in diameter; these were found to be very rich in iron and the cells containing them were regarded as analogous to "heart-failure cells". Other, less common, cells contained irregular granules of what was apparently carbon. Among the other granules in some of the cells were transparent, refractile, and apparently crystalline particles of uncertain nature. No particles were found which could be identified as beryllium oxide. Spectroscopic analysis showed that the lungs contained about 10 μg. of beryllium per 100 g. of dried lung. The authors postulate a specific sensitivity in the patient, as a number of his fellow workers, exposed to the same risk, were unaffected. *John Crofton.*

Diffuse Pulmonary Granulomatosis in Young Women Following Exposure to Beryllium Compounds in the Manufacture of Radio Tubes. Report of Five Cases. SLAVIN, P. (1949). *Amer. Rev. Tuberc.*, **60**, 755.

Five cases of chronic lung disease in young women are fully reported. All had been exposed to beryllium-containing dust while working in radio tube factories. The illness had an insidious onset, the chief symptoms being dyspnœa and cough; it ran a progressive course with no remissions. Radiography revealed miliary and discrete nodules and "connective tissue infiltrates", with calcification late in the disease. Multiple bleb formation was seen in all cases, and a cavity was present in one case. Spontaneous pneumothorax occurred in 2 cases. Microscopical examination of the lung granulomata showed a picture similar to that of sarcoidosis, with giant cells and concentrically arranged fibrous tissue which sometimes underwent hyaline change; there was no caseation. Unlike the condition in silicosis, massive conglomeration did not occur.

Two of the 5 patients were dead at the time of the report 2½ and 2 years respectively after the first symptoms; in one the symptoms had developed 3 years after the exposure to beryllium dust, which had been for 3 months only. The patients who survive have had symptoms for from 15 months to 10 years. *L. M. Franklin.*

Studies on the Respiratory Gas Exchange in One Hundred Anthracite Coal Miners with Pulmonary Complaints. MOTLEY, H. L., LANG, L. P., and GORDON, B. (1950). *Amer. Rev. Tuberc.*, **61**, 201.

The authors studied the respiratory gas exchange in a group of 100 men, all of whom had a long history of exposure to dust in anthracite mines, and all of whom complained of pulmonary symptoms: except that cases of proved pulmonary tuberculosis, cardiac decompensation, carcinoma of the lung, and other disabling medical conditions were rejected, they form an unselected consecutive series. Dyspnœa, cough, expectoration, weakness, loss of weight, pain in the chest, and hæmoptysis were the major symptoms, in that order of frequency. Samples of blood were obtained from the brachial artery in each case during rest and again immediately after a simple stepping-up exercise: the oxygen and carbon-dioxide content of these samples was determined, as well as that in the inspired and expired air. One-third of the men had no significant emphysema as judged by the relation of the volume of residual air to the total lung volume: one-third had advanced emphysema. The oxygen saturation of the arterial blood at rest bore little relation to the degree of impairment of respiratory function, but exercise commonly resulted in a significant decrease which, on the whole, was proportional to the degree of emphysema present.

Several factors are concerned in the disability resulting from anthracosilicosis, the commonest and most important of which is unequal alveolar ventilation; increased difficulty of diffusion across the alveolar membrane seems to play only a minor or occasional part. Polycythæmia or a raised level of hæmoglobin are rare findings even in the presence of advanced emphysema, reduced ventilatory capacity, arterial oxygen unsaturation, and marked dyspnœa on exertion.

The authors conclude that an accurate evaluation of the degree of impairment of pulmonary function requires measurements of ventilation efficiency, residual air, and oxygen uptake from the inspired air, and examination of arterial blood during rest and exercise. In some cases measurements of the pulmonary circulation are necessary. Because of the wide individual variations found in these determinations, no single test can be used for the satisfactory appraisal of disability. *H. E. Harding.*

The Use of Bronchodynamic Aerosols in the Study of Respiratory Function in Silicosis. PARMEGGIANI, L., and PINEROLO, A. (1949). *Med. d. Lavoro*, **40**, 297.

Attempts have been made to assess respiratory efficiency with the aid of aerosols, and some authorities claim that by this means an exact estimation of invalidity and incapacity for work is possible amongst operatives in dusty occupations suffering from respiratory in-

sufficiency. The classification of such patients into four groups is suggested: (1) those with latent respiratory embarrassment; (2) those with dyspnœa not greatly preventing activity: (3) those with serious physical limitations; and (4) those with grave respiratory disorders. For this purpose tests are carried out with aerosols of substances causing bronchial constriction and with others causing dilatation. The authors have studied the value of this method, using the method of Tiffeneau, with a Benedict spirometer and a Rainal nebulizer. Bronchoconstriction was obtained with 1% acetylcholine and bronchodilatation with 0·5% "iludrin" (hydroxyphenylethanol*iso*propylamine sulphate).

Of 80 subjects tested 36 had silicosis (with various degrees of disability), and 20 asthma, and 24 were normal controls. Under basal conditions of rest, with the patient fasting, the function of the lungs was fully investigated by spirometry before and after the administration of the aerosols, vital capacity, tidal air, ventilation per minute, and other values being recorded. Variations in the pulse rate and blood pressure were not very great during the experiments. The character of the breathing altered considerably in some subjects, notably in the asthmatics, in whom acetylcholine administration caused harsh, high-pitched breathing, or whistling sounds audible at some distance. When iludrin was used breathing immediately became easier. Somewhat similar effects were noted in silicotic subjects, whereas in normal subjects acetylcholine was generally well tolerated.

The experiments brought out clearly the influence of vegetative nervous factors on respiratory efficiency. It was concluded that the method used is not of much value as a means of assessing capacity for work in asthmatic subjects, since the personal factors, notably the degree of collaboration of the patient, vary greatly and cannot be predicted. This view is, however, modified in respect of silicotic subjects, who suffer from an organic and progressive disability less subject to nervous influences. Standardization of the tests is difficult. *G. C. Pether.*

The Fate of Oil Particles in the Lung and their Possible Relationship to the Development of Bronchiogenic Carcinoma. SANTE, L. R. (1949). *Amer. J. Roentgenol.*, **62**, 788.

Highly refined vegetable oils free from fatty acids are removed from the alveoli by phagocytic action and this leads eventually to complete removal without evidence of damage to the lung structures. Oil that has deteriorated should not be injected into the bronchial tree because of the possibility that fatty acids may be present. A change in colour of the oil is evidence of deterioration.

Animal oils contain a high percentage of fatty acids, and are thus in themselves irritant. Further, they remain longer in the alveoli and thus large amounts of fatty acids are formed. As a result lipoid pneumonia develops and may be fatal.

Mineral oils are also removed from the alveoli by the action of phagocytic cells but, being inert, they cannot readily be autolysed by the cells and are carried with little change through the regional lymphatics to the hilum, where they excite an extreme degree of cellular reaction which results in fibrosis and scar tissue. The phagocytic cells themselves are often destroyed by the oil which they contain. The resultant mass of paraffin oil droplet enmeshed in heavy scar tissue gives rise to a paraffinoma. Paraffinomas may attain several centimetres in diameter and are usually found about the large bronchial branches near the root of the lung at the site of the hilar lymph nodes.

The author discusses the possibility of carcinomatous change supervening in the paraffinomas and quotes one case in which this appears to have occurred. [The second case quoted is less convincing in that the lipoid change is distal to a primary bronchial carcinoma. It is well known that such change may occur in the absence of any history of inhalation of oil.] *L. G. Blair.*

Conditions for the Development of Cancer of the Lung. WASSINK, W. F. (1948). *Ned. Tijdschr. Geneesk.*, **4**, 3732.

A study of 143 cases of pulmonary cancer, 136 in men and 7 in women, was made from the points of view of occupation and tobacco-addiction. This form of cancer was found to occur chiefly among intellectuals and white-collar workers and seldom in agricultural workers. This group showed the highest degree of tobacco-addiction found among cancer sufferers. Similar but less definite figures were found for cases of endolaryngeal cancer. It is concluded that smoking and the inhalation of smoke-laden air constitute the foremost exogenous causal factors in the incidence of pulmonary cancer in men in the Netherlands. *A. Schaberg* (*Excerpta Medica*).

ENVIRONMENT

Health of Workers Exposed to Galena. BELDEN, E. A., and GARBER, L. F. (1949). *J. industr. Hyg.*, **31**, 347.

Commercial lead mining has been carried on in Missouri since 1732. The mineral is galena or lead sulphide with small amounts of zinc, silver, and some rarer metals. Sixteen men in four different mills who handled concentrates which contained 68 to 75% lead were examined clinically. Blood counts and estimations of lead in blood and urine were made. Stippled cell counts and basophilic aggregation tests gave values that were within normal limits. Analysis of blood and urine for lead revealed little abnormality except a rise during the week in the lead content of urine from 0·033 mg. to 0·159 mg. per litre. In blood amounts ranged from 0·003 to 0·142 mg. of lead per 100 g. of whole blood. Atmospheric samples collected over periods of 5 days during the summer season and 6 days during winter showed concentrations of lead sulphide varying from 2 to 50 times the accepted safe limit of 1·5 mg. of lead per 10 cubic metres of air. There were no cases of lead poisoning. *K. M. A. Perry.*

Health Hazards in Radiation Work. INGRAM, M. (1950). *Science*, **111**, 103.

This paper from the University of Rochester, Rochester, New York, deals with the dangers associated with ionizing radiations, all types of which produce similar biological effects in the form of varying degrees of cell injury.

The various types of radiations are considered. Properties of *x* rays are similar to those of light waves, but they are more penetrating. Protection is usually adequate, but very high voltage *x*-ray machines must be housed in separate buildings to avoid irradiation of occupants of nearby rooms. Gamma rays occur in association with both naturally occurring and artificially produced radioactive elements and are also produced by high voltage *x*-ray machines, accelerators, and chain-reacting piles. The particulate alpha radiation is strongly ionizing but only slightly penetrating. Once an alpha-emitting isotope is in the body a certain proportion is more or less permanently deposited in some tissue, especially bone. There is no efficient method for hastening its removal, hence great damage is done. Certain isotopes of elements heavier than lead, for example, radium, plutonium, and polonium, are alpha emitters. Neutrons are uncharged particles and have great penetrating power. The hazards are associated with the operation of cyclotrons and chain-reacting piles and with investigations in which neutron sources are used.

The seriousness of the hazard encountered in work involving any type of radioactive isotope is dependent in part on the half-life of the isotope. The slowness to recognize radiation hazards is emphasized. It is stressed that any effect radiation has on normal tissues is harmful. Tolerance doses have been arrived at from studies designed to determine how much radiation can be received without producing any detectable damage or how little radiation may be received and yet produce definite changes, immediate or remote.

In the determination of safe working conditions information is required about the type and the intensity of the radiation, and also about the nature of the exposure. Frequently repeated exposure to low doses constitutes the greatest hazard. A limited number of exposures to a large dose is met with in patients receiving radiotherapy, and occasional gross over-exposure occurs in experimental work. An appreciation of the biological effects of radiation is necessary. Animal experiments have revealed that all tissues do not respond in the same way to given amounts of radiation. Skin, white blood cells, bone marrow, reproductive cells, and cells lining the intestines respond readily to small doses of radiation. Adult erythrocytes, bone, and nerves, are more or less resistant. Hæmatological studies are one of the important methods of detecting radiation damage, especially in total body irradiation. Local irradiation may produce local changes. The possibility of the occurrence of alteration in fertility and genetic change in man remains speculative, but is obviously an important consideration in defining safe levels of exposure.

The accepted standards of safety are in the nature of approximations, requiring revision in the light of new knowledge. It is necessary to recognize the hazards, define safe working conditions, and maintain them. A certain distance should be kept between the worker and the source of the radiation. In most cases the distance can be shortened by interposing a shield of a substance which readily absorbs the radiation.

Treatment of severe radiation injury is considered. Health protection of radiation workers has been remarkably successful in atomic energy installations.

Edward M. McGirr.

Lesions of the Eye from Radiant Energy. COGAN, D. G. (1950). *J. Amer. med. Ass.*, **142**, 145.

The two main types of radiant energy are : (1) Electromagnetic : wireless, diathermy, infra-red, visible, ultraviolet, Grenz, roentgen, gamma, and cosmic rays ; (2) Corpuscular : electrons (beta and cathode rays) and neutrons. The biological effect of the various forms depends on their power of penetration and absorption. The wireless and long diathermy waves are highly penetrative and unabsorbed, and therefore have little biological effect. Shorter diathermy waves heat and have a potential effect on the anterior segment of the eye. The infra-red rays are largely absorbed by the cornea and iris, but the shorter ones penetrate the media, to be similarly absorbed in the retinal pigment. Of the visible rays 70% of those in the red end of the spectrum reach the retina, while only 10% of the violet rays can penetrate so far. In the ultraviolet band the longer rays may penetrate to the lens, but the shorter will all be absorbed by the cornea. Grenz rays and beta and cathode-ray particles have only small penetrating power (99% are absorbed in the first millimetre of thickness). The penetration of the roentgen and gamma rays is proportional to the potential of their generation.

In assessing the harmful effects, certain factors must be considered. Maximal damage will be found at the site of absorption of a given ray. Most sources of energy emit a mixed waveband. The longer the wave-length, the shorter will be the latent period. The vulnerability of a tissue is in inverse proportion to its reparative power.

On these lines, the author deals with the main ocular structures and briefly describes the known lesions induced by various wave-bands. The lines on which industrial injury must be combated are by widespread mechanization of dangerous processes and the use of suitable screens where mechanization is not practicable.

J. E. M. Ayoub.

GENERAL

Fatalities Resulting from Exposure to Simulated High Altitudes in Decompression Chambers. A Clinico-pathologic Study of Five Cases. HAYMAKER, W., and DAVISON, C. (1950). *J. Neuropath. exp. Neurol.*, **9**, 29.

It is certainly remarkable, as the authors of this paper point out, that there should have been so few fatalities amongst the thousands of airmen who, during the war, entered decompression chambers for instruction in the hazards of flying at high altitudes and in the use of oxygen apparatus. In these decompression chambers

the barometric pressure was lowered to simulate altitudes of between 30,000 and 38,000 feet (9,000 to 11,400 m.). Oxygen was given as a routine when a simulated altitude of 10,000 feet was reached. The authors were able to trace only seven deaths resulting from exposure to these simulated high altitudes and five of these form the subject of this detailed study.

The ages of 4 of the victims were between 22 and 26 years; the fifth was 38 years old. All were in perfect health before the tests. Other men in the same decompression chambers exposed to similar conditions suffered no ill effects. The only factor common to the fatal group was adiposity or a tendency thereto. Symptoms developed during the tests in all the fatal cases, the time of onset varying from 5 minutes to just over an hour after the tests had begun. "Bends" and "chokes" occurred in only 2 cases. In one case the symptoms were solely those of shock. In the others, such neurological manifestations as giddiness, diplopia, ocular palsies, monoplegia, and hemiplegia developed. Death occurred from 8½ hours to 3 days 10 hours after the onset of symptoms. At necropsy, the changes in the viscera were those associated with shock, namely, the signs of peripheral vascular collapse, with pleural effusions and œdema of the lungs. Fat emboli were present in the lungs in 4 cases. The authors consider that these fat emboli came from the bone marrow, nitrogen bubbles forming in the venous channels causing rupture of their walls and so allowing free fat to pass into the circulation. In the brain there were ischæmic changes in the nerve cells of the cerebral cortex, especially in the deeper laminæ in the depths of the sulci. Ischæmia was particularly obvious in Sommer's sector of the hippocampus. The meninges were moderately congested in all cases. An interesting observation was that of perivascular gliosis in the cortex. This rapid reaction of astrocytes is somewhat surprising. Early perivascular demyelination in the sub-cortical white matter was also noted, and in one case fat emboli were seen in the vessels of the white matter. Sections of the spinal cord were available in only 2 cases. In one of these the cord was normal; in the other many small perivascular hæmorrhages were present in both the grey and white matter, whilst the grey matter was œdematous and disintegrated. These changes in the cord were similar to those found in caisson disease.

The authors conclude that the cause of death in these cases is aero-embolism, which leads to fatal shock. As no one part of the nervous system is involved more than another, they suggest that it is a matter of chance where the nitrogen bubbles lodge, and that if they lodge in sufficient quantities in vital centres death will ensue.

Ruby O. Stern.

Hearing of Airline Pilots. A Ten-year Study. SIMONTON, K. M. (1949). *Aviat. Med.*, **20**, 418.

Over a period of 10 years, the hearing of 47 civil-airline pilots was tested periodically by threshold audiometry. The author admits that the tests were carried out under conditions in which not inconsiderable errors could occur. Significant average loss of hearing was found only at frequencies of 4,096 and 11,584 cycles per second; at 4,096 cycles the average loss was 10·3 decibels, and at this frequency a loss exceeding 25 decibels was found in 14 ears. Loss of hearing was in some individuals unilateral. Analysis of the results suggested that pilots whose initial audiogram showed a descending curve in the high-tone frequencies were more liable to have greater subsequent loss of hearing than those with other types of audiogram.

As changes in hearing over the 512- to 2,048-cycle frequency range were insignificant, the author concludes that loss of hearing due to flight is not a serious problem.

[The author's findings hardly justify this conclusion unless individual results be rejected in favour of averages, and changes outside the 512- to 2,084-cycle range be ignored. Moreover, the results of audiometry are suspect; although the subjects were 10 years older, hearing at 11,584 cycles per second was better in 26 out of 29 ears.] *T. A. Clarke.*

Bacterial Contamination of Air in Underground Trains. WILLIAMS, R. E. O., HIRCH, A., and LIDWELL, O. M. (1950). *Lancet*, **1**, 128.

Surveys of the bacterial flora of the air, by means of a special type of slit-sampler for long period sampling, have recently been undertaken in underground trains running on the Northern Line between Edgware and Morden. It was shown that even during the rush hours of the evening, when more than 100 passengers crowded into each car for certain parts of the journey, the maximum total viable bacterial count in any one-minute period was only 170 per c.ft. (6,000 per c.m.) of air, with an average for one route of 47 per c.ft. (1,660 per c.m.). The streptococcal counts were extremely low; the average for the route quoted above was only 0·33 per c.ft. (11·6 per c.m.). These figures compare very favourably with the mean counts obtained in a large series of observations in elementary school class rooms. Whenever a train stopped at a station, the movement of the passengers entering or leaving the car appeared to cause an immediate rise in the colony count, but this tended to settle very quickly soon after the train got under way—even when the car was most crowded. As would be expected, there was a steady rise in temperature of about 12° F. (6·7° C.) during each run [presumably during the long underground section] but there was no systematic change in the relative humidity.

The slit-sampler itself was housed in a driver's cab forming part of the central car of the train, and was fed with air drawn from the end of the passenger car (a "non-smoker") by means of a fan over which was a closely fitting hood with its orifice projecting into the passenger car. Observers at each end of the car kept records of the number of passengers per section, and the temperature and humidity were measured at about 10-minute intervals on the excess air from the sampler hood. The authors appreciate the possibility that their figures might not apply to air drawn from the centre of the car. But it is clear, within the limit of these experi-

ments, that there was no massive accumulation of bacteria in the air of the underground trains in question.
H. J. Bensted.

Triethylene Glycol in Air Disinfection. GEDDA, E. (1949). *Nord. Med.*, **41**, 363.

Two series of tests were carried out to determine the efficacy of vaporized triethylene glycol for the disinfection of air in public places. One of the places was a waiting room for out-patients at the Ear, Nose and Throat Clinic, Sahlgren Hospital, Gothenburg, with a volume of about 400 cubic metres and of very irregular contour; and the other a nursery at Vidkärr Children's Home, Gothenburg.

In the former series similar control tests were made in the same waiting room for the same number of days, but without the use of glycol; in the latter series a control room identical in every respect with the rest nursery was used.

In the waiting-room tests the bacterial concentration in the air was reduced by about 30% by use of triethylene glycol, and at the children's home a reduction of about 40% in the test nursery compared with the control room was obtained. The difference in each case is statistically significant.

The atomizer employed was a modified "aromator" electrically heated and fitted with an electric fan. It provided sufficient but inconstant concentrations in the peripheral parts of the waiting room. For the bacterial count a Bourdillon apparatus was employed, colonies in blood-agar cultures being counted, without differential calculation of the micro-organisms, after 24 hours in a thermostat at 37° C. The relative humidity ranged from 35 to 60% in the first series and 70 to 80% in the second. The average bacterial count in control rooms was 66 per 100 litres of air in the E.N.T. clinic (23 tests a day for 5 days) and 324 per 100 litres in the children's playroom (17 tests a day for 6 days). The concentration of glycol in air aimed at was 0·005 mg. per litre.
A. M. M. Wilson.

The Rate of Bactericidal Action of Triethylene Glycol Vapor on Microorganisms Dispersed into the Air in Small Droplets. LESTER, W., ROBERTSON, O. H., PUCK, T. T., and WISE, H. (1949). *Amer. J. Hyg.*, **50**, 175.

Freshly atomized triethylene glycol has a rapid bactericidal action, which is difficult to measure accurately as the chemical rapidly condenses on surfaces in the experimental chamber, thus reducing the concentration in the atmosphere. By means of a special apparatus whereby the concentration of triethylene glycol and water vapour could be maintained constant in the atmosphere, the bactericidal action of glycol vapour at various degrees of saturation and under varying conditions of humidity was measured. With a relative humidity of between 5 and 50% the bactericidal action rose progressively with the concentration of glycol vapour, but as the relative humidity was raised above 50% there was a rapid diminution of bactericidal action at all concentrations of glycol vapour.
Scott Thomson.

Boils. An Epidemiological Survey. WHITWELL, G. P. B., and SUTHERLAND, I. (1950). *Brit. J. Derm. Syph.*, **62**, 109.

The incidence of boils in an industrial community increased during the period 1943–7, with peaks in October or November. The site of boils (including styes and carbuncles) was studied in this predominantly male community during 1946 and 1947. The commonest sites were the face, head, and neck, followed by the forearms and wrists. Gluteal boils accounted for less than 1%. Whereas 26% of boils in men were on the neck, only 4% in women were on that site. Most of them were in the lower occipital region, well above the collar line. It is suggested that staphylococcal contamination of the skin depends on spread from the nose by the fingers.
E. Lipman Cohen.

Industrial Efficiency as Affected by Food Intake During Mid-morning and Mid-afternoon Rest Periods. HALDI, J., and WYNN, W. (1949). *J. appl. Physiol.*, **2**, 268.

The common inclination of workers to stop work in the mid-morning and mid-afternoon and to take food or drink may be due to a physiological need for food or to a psychological need for a restful change from monotony. The efficiency of women working all day at top speed sewing cotton bags was therefore studied in relation to the kind and amount of food taken at mid-morning and mid-afternoon rest periods. All workers sewed at top speed because they were paid according to the number of bags sewn.

The rest periods were of 15 minutes, and during a period of 3 weeks the workers received at this time either: (1) sausage, chips, bun, and soft drink (650 Calories); or (2) cake, banana, milk (300 Calories); or (3) milk chocolate (150 Calories); or (4) soft drink (80 Calories). On 2 days they were given nothing. The differences in output on various days were not statistically significant. It is therefore inferred that such supplements are not required as far as provision of energy is concerned. It is, however, pointed out that the workers were by no means pleased at being allowed no food or drink on the two control days, and it is therefore thought advisable to allow workers a wide choice of food or drink during rest periods.
S. S. B. Gilder.

The Iron and Steel Industry. SWANSTON, C. (1950). *Lancet*, **1**, 191.

The passage of the Iron and Steel Act of 1949, providing for the nationalization of the main output of the industry in Great Britain, has prompted the author to present this comprehensive survey of the different processes within the industry and the health hazards to which the workers are exposed. She traces the history of the industry and the reasons for its present establishment in concentrated areas of South Wales, the north-east coast, Lanarkshire, the west of Scotland, and the Midlands. The 1931 depression caused great unemployment, and the setting up of the Iron and Steel Federation in 1933 was necessary

to stop the industry's decline. Reorganization, with the establishment of new plants in distressed areas, did much to relieve the situation and was of the utmost importance in meeting the sudden increase in demand during the recent war. Recommendations to bring plant up to date, provide additional furnace capacity and rolling mills, and modernize iron foundries were made in 1945 by the Federation and the Joint Iron Council as a means of increasing production and overcoming the serious shortage of labour. Although it is difficult to differentiate between the iron and steel industry and the metal and engineering trades, the former is essentially concerned with the manufacture of steel ingots, plates, and castings and the production of iron castings. The initial process is the production of pig-iron from the smelting of iron ore, and a description is given of the working of a blast furnace. The various processes used in the manufacture of steel—the Bessemer, the open-hearth, the crucible, and the electric-arc furnace—are also described, and attention is drawn to the health hazards involved. The physical demands of the industry are heavy and are not improved by the environmental conditions under which some of the work has to be performed. Recommendations made in 1947 by the Joint Advisory Committee included: (1) provision of better amenities and more comfortable and healthy working conditions; (2) improvement in the appearance of iron foundries; and (3) improvement in atmospheric conditions by prevention or removal of dust, smoke, and fumes.

Compared with other industries the accident risk is significantly high, particularly in respect of accidents due to handling goods, to gassing, and to molten metal or corrosive substances. Certain industrial diseases are especially frequent in the iron and steel industry. In 1937 the industry was responsible for 82% of cases of industrial cataract and 100% of cases of nickel-carbonyl poisoning notified, while tenosynovitis of the wrist, "beat hand", "beat knee", and "beat elbow", which are predominantly diseases of miners, are found to a moderate extent in heavy metal workers. Carbon monoxide gassing is a constant source of danger in blast furnaces, and the smelting of metals may also produce the hazards of carbon dioxide, sulphur dioxide, hydrogen sulphide, and, occasionally, arsenious oxide poisoning. Steel dressing and the cleaning of castings with hand tools produces a dust of silica particles from the sand in the moulds, and, until a harmless substitute is found for making moulds, the risk of silicosis will continue. Sand blasting used to constitute the chief silicosis hazard, but this has been greatly reduced by replacement of the sand by steel shot. There is an increased liability in the industry to respiratory and rheumatic diseases, due probably to the environmental factors of rapid changes of temperature and exposure to weather, and to the heavy nature of the work, as well as to the irritant matter inhaled in the form of dust and fumes. Many occupations in this industry show significantly high death rates from cancer of the lungs and respiratory passages, and the author suggests that the frequent breathing of hot air and gases may, in itself, be harmful and capable of producing tissue changes.

A. Lloyd Potter.

Correction.—Dr. Buckell writes: "I am very sorry an error has got through all our checking. In my paper on 'The Toxicity of Methyl Iodide' [July, No. 3, Vol. 7] line 43 (first column) page 123 should read '0·15–0·22 *grams* per kg. body weight.'"

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